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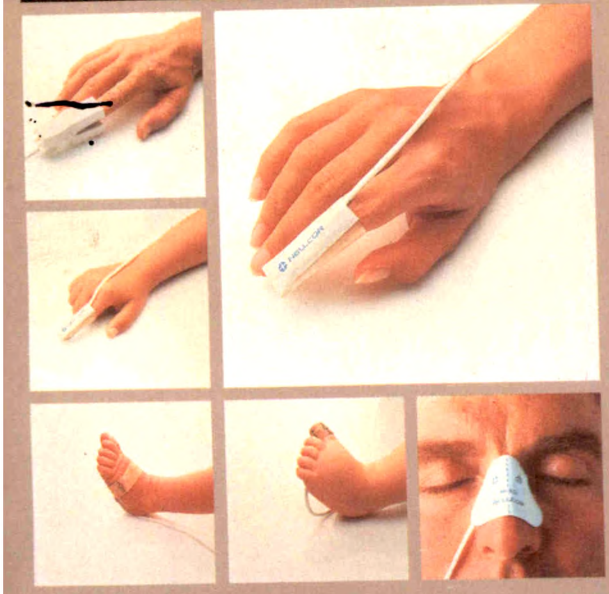

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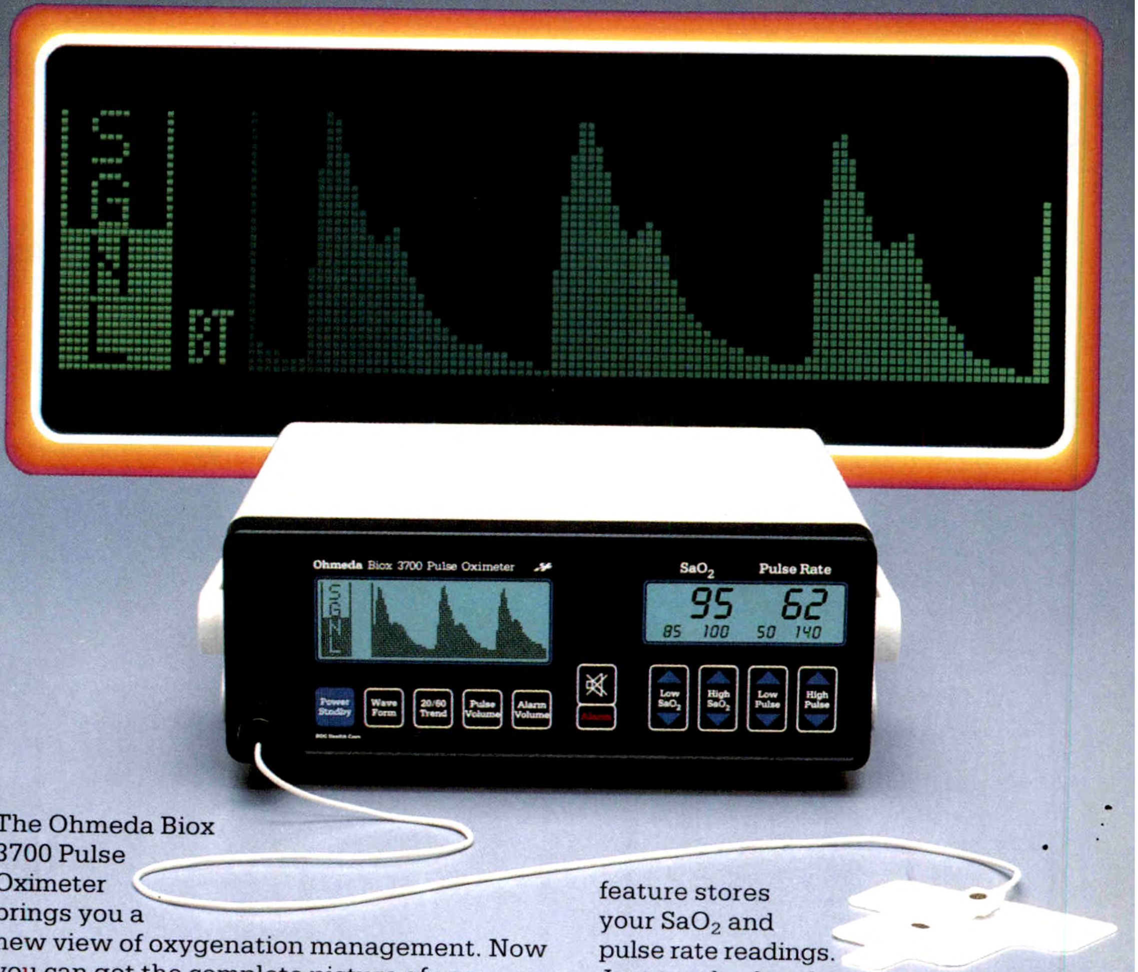

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The Advantage of Pulse Oximetry as a Monitor.

J. F. Nunn, Ph.D., M.D., F.R.C.S., F.F.A.R.C.S.;
Division of Anaesthesia, Clinical Research Centre, Middlesex, UK

Our starting point in consideration of instruments to measure the state of oxygenation of a patient must be recognition of the unreliability of the unaided human senses and particularly in the detection of cyanosis. Intermittent monitoring of arterial PO_2 is little better because it is usually undertaken on discrete samples, while hypoxaemia is often episodic and rapid in onset. It is the transients which are important in, for example, sleep apnoea, post-operative obstructive apnoea and dental anaesthesia. Furthermore, monitoring of arterial blood is invasive and people are naturally reluctant to use it in routine situations, such as dental anaesthesia in the chair. Delay in analysis reduces its value in emergency situations.

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This article is an excerpt from **Pulse Oximetry** edited by: J. P. Payne and J. W. Severinghaus.
Publishers: Springer-Verlag, Berlin, Heidelberg, New York, London, Paris, Tokyo.

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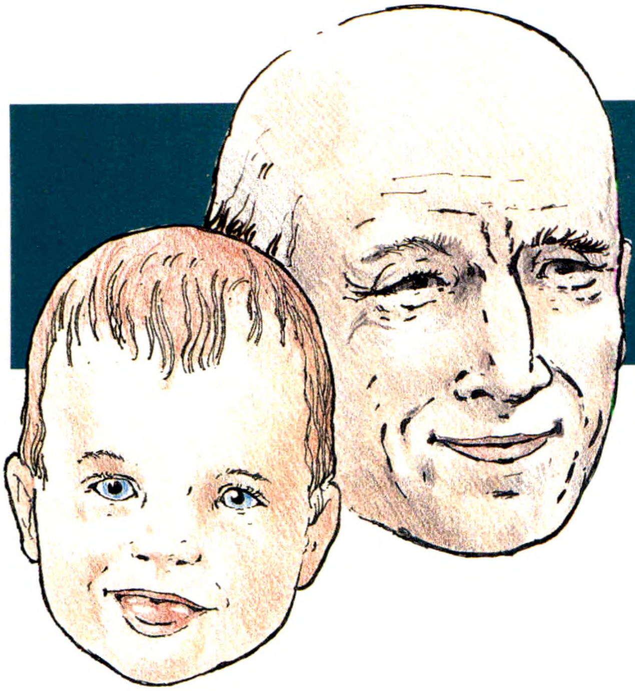

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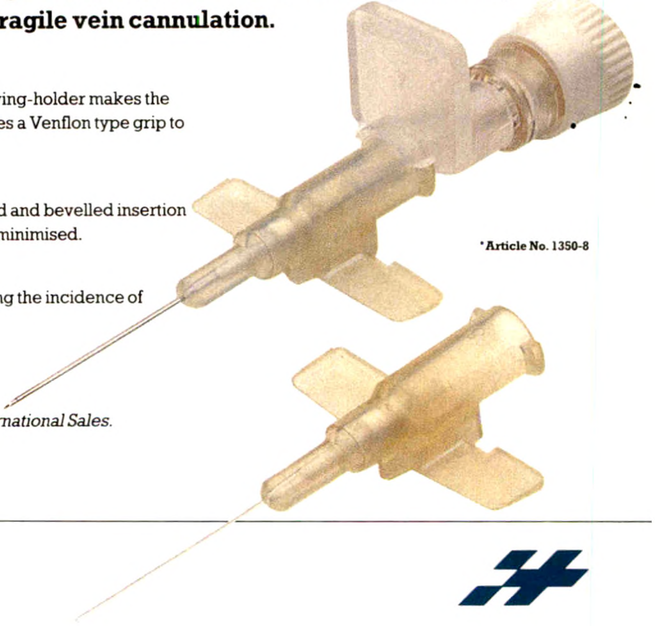

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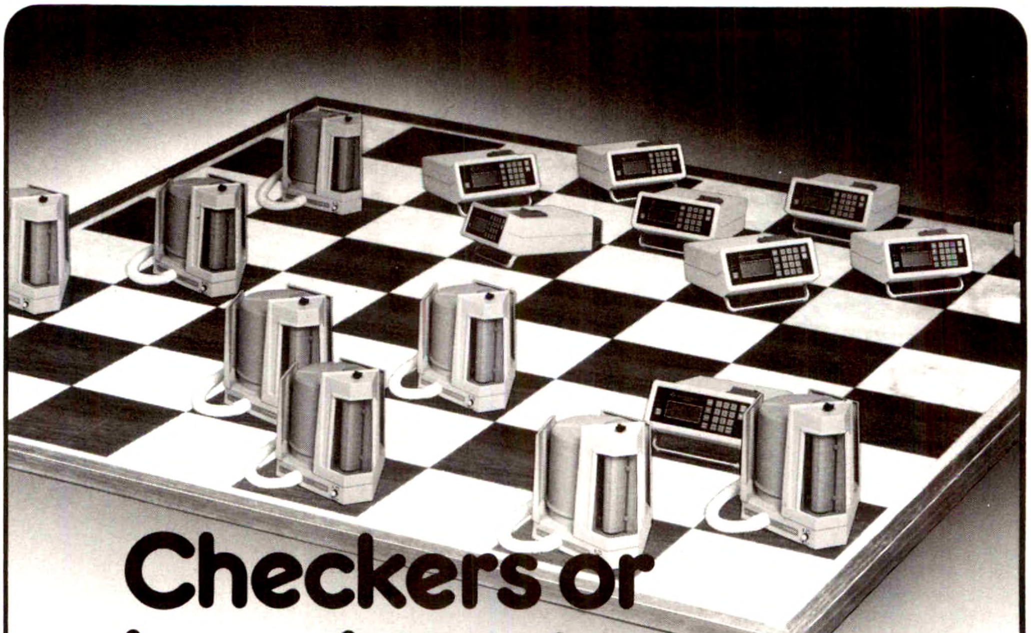

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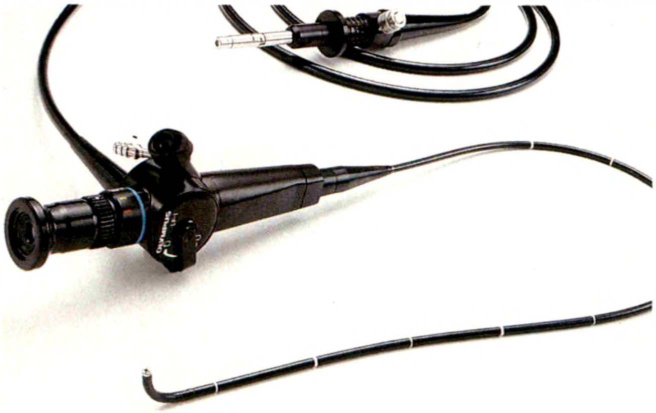

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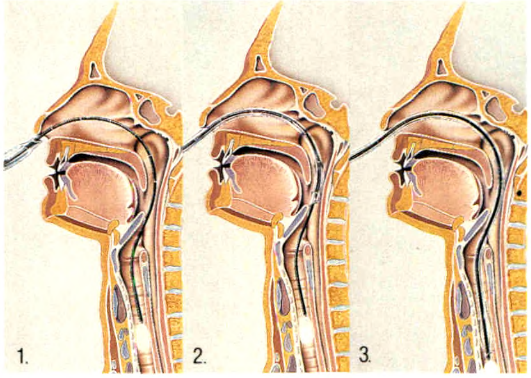

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★ 1. Fiberoptic bronchoscopy for anaesthesia. Watson, C.B., Anaesthesiology Review (1982), IX: 17-25.
2. Use of flexible fiberoptic bronchoscope to change endotracheal tubes in critically ill patients. Rosenbaum, S.H., Am. Soc. Anaesthes. (1981), 54: 169-170.
3. Endotracheal intubation with the flexible fiberoptic bronchoscope. Davidson, T.M. Anaesthesiology Review (1975), 54: 346-349.
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Anaesthesia, 1987, Volume 42, pages 1–2

Editorial

Smoking and anaesthesia

About one-third of adult patients who present for surgery are cigarette smokers. The evidence is overwhelming that they have an increased operative risk.[1–3] A period of smoking abstinence before surgery can lessen this risk. Smoking has wide ranging effects on normal physiology, and on smoking cessation these are reversible to a greater or lesser extent. How long then should we advise patients to stop smoking before surgery? In order to answer this question the principal pathophysiological effects of smoking need to be briefly reviewed.

The precise cause of the deleterious long-term cardiovascular hazard of smoking has not been defined, but there are clear acute cardiovascular effects of smoking. Nicotine is a potent adrenergic agonist and increases heart rate and arterial blood pressure, significantly increasing peripheral vascular resistance. In addition, heavy smokers may have up to 15 percent of the available oxygen binding sites of haemoglobin occupied by carbon monoxide, which results in an approximately 25 percent reduction in available oxygen. Thus, nicotine increases myocardial oxygen consumption whereas carboxyhaemoglobin (CoHb) reduces myocardial oxygen supply. Recently, smoking has also been convincingly demonstrated to have a marked influence on coronary vascular resistance, especially at the site of atheromatous stenosis.[4] The clinical corollary of these three observations is that smoking worsens the symptoms of angina, whereas stopping smoking is associated with a clear improvement in cardiovascular fitness.[5] In terms of improved oxygenation, therefore, we are amply justified in seeking to persuade our patients to stop smoking before surgery and, because of the relatively short half-life of CoHb, a period of abstinence of as little as 12 hours improves available oxygen. This would be of benefit to all patients presenting for anaesthesia and surgery, but is of greatest importance to those with coronary artery disease.

A six-fold increase in the postoperative respiratory morbidity in patients smoking more than ten cigarettes a day was reported by Morton in 1944;[1] numerous studies have confirmed Morton's original observation. The contribution of anaesthesia alone to the pathophysiology of pulmonary complications is not clear, although recent work suggests that inhalational agents, such as halothane, may cause deleterious effects on pulmonary macrophage function; this leads to an increased inability to clear material.[6] The pre-operative effects of smoking are additional to any effects of anaesthesia. Smoking also has potent influences on several aspects of the immune response including reductions in neutrophil activity, in immunoglobulin concentrations, and natural killer cell activity. Many aspects of immune function return to normal after six weeks' abstinence and there is some improvement in tracheobronchial clearance and reversible small airways disease. Therefore, to make any significant impact on postoperative respiratory morbidity, a minimum of six weeks' abstinence is necessary.

Smoking is a dangerous addiction. A middle-aged heavy smoker runs about as much risk of dying in the following 12 months as a non-smoker ten years older. Smokers also have about one-third more time off work than non-smokers. Advice to stop smoking given in a hospital and reinforced in follow-up clinics has been shown to be successful in persuading people to quit the habit.[7] Considered, sensible advice by anaesthetists and their surgical colleagues could therefore help towards significantly decreasing the risks of surgery in a substantial proportion of the population.

The Association of Anaesthetists of Great Britain and Ireland is collaborating with the Health Education Council in order to provide advice for pre-operative smoking patients. This will comprise information for anaesthetists and for surgeons and probably nurses. A patient's leaflet will explain the risks of smoking before an operation, emphasising the benefits of giving up and providing information on where to get assistance. A trial of the effectiveness and compliance will be undertaken in test areas.

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Smoking education has the highest priority at the Health Education Council. If an effective campaign can be mounted, the Association of Anaesthetists will have decreased the risks of surgery in a substantial proportion of the population and will at the same time have contributed to the increasing decline in smoking prevalence.

Guy's Hospital, R.M. JONES
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Editorial notices

Submission of manuscripts

Manuscripts must be submitted in accordance with the internationally recognized *Uniform requirements for manuscripts submitted to biomedical journals* (*British Medical Journal* 1979; **1:** 532–5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

News and Notices sections

The first issue of each quarter will contain full News and Notices sections.

Copy dates for these will be: 13 March for July 1987, 12 June for September 1987 and 11 September 1987 for January 1988.

Supplementary News and Notices sections will be published in other issues but they will contain only material which requires publication before the next full News and Notices section.

Anaesthesia, 1987, Volume 42, pages 3–6

Propofol for intravenous sedation

N. MACKENZIE AND I. S. GRANT

Summary

This study investigated the properties of propofol when given by subanaesthetic infusion to provide sedation as an adjunct to spinal anaesthesia for lower limb surgery in 40 patients. Sedation, defined as sleep with preservation of eyelash reflex and purposeful reaction to verbal or mild physical stimulation, was satisfactorily achieved and maintained with minimal complications. The mean duration of infusion was 98 minutes and a mean infusion rate of 3.0 mg/kg/hour was required in patients over 65. This was significantly less ($p < 0.005$) *than the 4.1 mg/kg/hour required in younger patients. Recovery was impressively rapid; patients regained full consciousness approximately 4 minutes after the end of infusion, and were free from minor postoperative sequelae. Conversion to general anaesthesia was achieved in three patients where surgery encroached outside the analgesic field of the regional block, simply by increasing the infusion rate to approximately 10 mg/kg/hour.*

Key words

Anaesthetics, intravenous; propofol.
Anaesthetic techniques, regional; spinal.

Regional anaesthesia is becoming an increasingly important aspect of anaesthetic practice. Its advantages include the avoidance of certain risks inherent in general anaesthesia, particularly those of airway obstruction and pulmonary aspiration, the avoidance of operating theatre pollution, the provision of good postoperative analgesia and the benefits in certain pre-existing medical conditions.[1] Most patients, however, express a preference for being asleep during the surgical procedure[2] and various methods have been described to provide this; these range from intravenous or inhalational sedation to full general anaesthesia. To avoid the disadvantages of the latter, light sedation with an intravenous agent is obviously the method of choice. However, to preserve the benefits of the local technique, recovery must be rapid and clear-headed, with freedom from minor postoperative sequelae.

Propofol, the new emulsion formulation of di-isopropylphenol, with its attractive pharmacokinetic profile,[3] would appear to be a logical agent to use in this manner. Previous studies with the Cremophor formulation showed that its use was associated with rapid recovery and freedom from postoperative side effects.[4,5] Indeed, two recent studies with the emulsion formulation have shown that propofol, when given either by intermittent bolus or continuous infusion, is an excellent agent for the provision of light general anaesthesia as an adjunct to regional blockade.[6,7]

There has been no work to date, however, on the use of propofol by subanaesthetic infusion

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N. Mackenzie, MB, ChB, FFARCS, Consultant, I.S. Grant, MB, ChB, FRCP (Ed), FFARCS, Consultant, Department of Anaesthetics, Ninewells Teaching Hospital, Dundee DD1 9SY.

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to provide sedation and this study was carried out to investigate this possibility further.

Methods

Forty patients of ASA class 1 and 2 between the ages of 16 and 78 undergoing orthopaedic surgery were studied. Each patient gave informed consent and the study was approved by the local hospital ethics committee.

All patients were premedicated with oral benzodiazepine. On arrival in the anaesthetic room an intravenous infusion of Hartmann's solution was started via a cannula in the dorsum of the hand, and baseline heart rate and arterial blood pressure were recorded. An infusion of a 1% solution of propofol was then commenced via an Injectomat syringe pump (Dylade Medical, Runcorn, Cheshire) into the intravenous line and spinal anaesthesia instituted with an intrathecal injection of 2.5 or 3 ml of 0.75% plain bupivacaine via a 25-gauge needle in the lumbar region. The patient was then prepared for surgery in the usual manner.

Heart rate, blood pressure and conscious level were assessed at 10-minute intervals; the latter used a five-point sedation score (Table 1). The presence of any side effects such as pain or discomfort at the injection site, respiratory problems, undue hypotension, excitatory phenomena or movement in response to surgery, was noted. The infusion rate was adjusted during surgery to maintain an appropriate level of sedation; this was classified as sleep with preservation of eyelash reflex and purposeful reaction to verbal or mild physical stimulation. All patients breathed air or oxygen-enriched air throughout; no further anaesthetic or analgesic drugs were given and the infusion was generally discontinued 5–10 minutes before the end of surgery.

Table 1. Sedation scores.

1	Fully awake and orientated
2	Drowsy
3	Eyes closed but rousable to command
4	Eyes closed but rousable to mild physical stimulation (earlobe tug)
5	Eyes closed but unrousable to mild physical stimulation

The times taken from the end of infusion for patients to open their eyes and become fully conscious and orientated were recorded and an overall assessment was made of the quality of sedation and ease of its control. Postoperative recovery was assessed 2–4 hours later and again at 24 hours; particular note was taken of the presence of side effects such as headache, drowsiness, nausea and vomiting. Patients were specifically asked about awareness during the surgical procedure and whether they would be happy to have the same anaesthetic technique again.

Results

Forty patients were studied, 23 female and 17 male. The mean age was 52.5 years (SEM 2.61) and weight 68.2 kg (SEM 2.01). The first 13 patients received an initial infusion rate of 4 mg/kg/hour for 30 minutes. All were well sedated by this time, sleeping quietly but rousable to command or earlobe tug, that is, corresponding to a score of 3 or 4 as shown in Table 1. The remaining 27 patients had a faster initial infusion of 6 mg/kg/hour for 10 minutes, reduced to 4 mg/kg/hour for the next 10 minutes, to achieve sedation more quickly. Once satisfactory sedation was established in both groups, it was maintained at this level by adjusting the infusion rate if necessary.

Sedation characteristics were good throughout, with no discomfort or pain on infusion. Excitatory phenomena, consisting of limb twitching, were exhibited transiently by three patients. These were mild and required no treatment. Airway maintenance was excellent, with no instances of obstruction, cough, laryngospasm, respiratory depression or apnoea. The blood pressure and heart rate changes that occurred were entirely compatible with spinal anaesthesia or operative blood loss and could not be attributed to the propofol infusion *per se*. Depth control was universally good and never excessively deep. Four patients required additional small boluses of propofol (25–50 mg). In three, surgery encroached outside the analgesic field of the spinal block and sedation was simply converted to general anaesthesia by increasing the infusion rate to approximately 10 mg/kg/hour and supplementing with nitrous oxide.

The average duration of surgery was 70 minutes and that of the infusion 98 minutes (Table 2). In no case did the combined regional

Table 2. Infusion data: mean (SEM).

	Mean	Range	
Duration of infusion, minutes	97.6 (10.47)	35–302	
Duration of surgery, minutes	70.0 (9.88)	13–280	
Start of infusion to surgery, minutes	33.3 (1.78)	20–65	
Start of infusion to spinal, minutes	10.1 (1.00)	2–35	
End of infusion to end of surgery, minutes	6.3 (0.68)	2–20	
End of infusion to eyes open, minutes	3.9 (0.49)	1–11	
End of infusion to return of full consciousness, minutes	4.4 (0.51)	1–11	
Mean infusion rate, mg/kg/hour	3.8 (0.125)	2.3–5.6	
$\leqslant 65$ years ($n=30$)	4.1 (0.12)	2.6–5.6	$p<0.0005$
>65 years ($n=8$)	3.0 (0.18)	2.3–3.5	

and sedation technique cause any hold up to surgery; the mean interval between commencing infusion and skin incision was 33 minutes. The majority of this time was surgically generated in scrubbing-up, gowning and preparing and draping the patient. The overall mean infusion rate was 3.8 mg/kg/hour but patients over 65 required significantly less than younger patients, at 3.0 and 4.1 mg/kg/hour, respectively. Two patients with a history of heavy alcohol intake required a substantially higher infusion rate of 5.6 mg/kg/hour.

The infusion was stopped, on average, 6 minutes before the end of surgery and most patients regained full consciousness within this period. This rapid recovery was impressively clear-headed and devoid of any early postoperative sequelae such as headache, confusion, restlessness, nausea or vomiting. A strong desire for food was a common feature among patients in this early postoperative period. The only side effects which occurred in the subsequent 24 hours were three cases of nausea and vomiting, all following intramuscular opioid analgesia once the spinal block had worn off. Two patients who received the slower initial infusion rate reported some early awareness of the operative procedure. All patients expressed willingness to have the same anaesthetic technique again and several volunteered how well they felt in comparison to previous experiences of general anaesthesia.

Discussion

Propofol by subanaesthetic infusion has been shown to provide excellent sedation as an adjunct to spinal blockade. The technique is safe, simple and versatile and causes no delay to surgery. Depth of sedation can be easily altered by adjusting the infusion rate and conversion to general anaesthesia is simple should the regional block prove ineffective. The technique is relatively free from side effects, in particular the respiratory and cardiovascular depression produced by propofol when given by higher infusion rates for general anaesthesia.[8] Recovery is impressively rapid, even in elderly patients, and devoid of side effects; full orientation generally returns within 5 minutes of stopping the infusion. The reduced dose requirement for the elderly is in keeping with previous findings related to propofol for induction of anaesthesia.[9]

With the resurgence of interest in regional anaesthesia,[10,11] the provision of good sedation becomes increasingly important if the advantages of the regional techniques are to be exploited to the full. Diazepam, and more recently midazolam, are the most widely used sedative drugs in anaesthetic practice.[2]. Although the venous complications of diazepam have been largely overcome with reformulation in an emulsion,[12] the problems of patient variability and prolonged recovery still remain with the benzodiazepines, particularly in the elderly.[13–17]

Althesin infusion was used successfully to supplement regional anaesthesia,[18] but this is now no longer available. Chlormethiazole infusion has also been advocated by some workers, but its long elimination half-life leads to prolonged recovery, so that patients can take up to 40 minutes to recover consciousness after the infusion is stopped.[19,20]

Methohexitone and propofol have been shown previously to be suitable agents for the provision of light general anaesthesia to cover surgery under regional blockade when given either by intermittent bolus or continuous infusion.[4,5] Involuntary movement was, however, a problem with methohexitone and recovery was also more prolonged, with a higher incidence of side effects.

This study has extended the clinical range of propofol into the field of sedation. The technique is safe, simple and versatile; a mean overall infusion rate of 3.8 mg/kg/hour provides excellent sedation with rapid, trouble-free recovery, bearing in mind the reduced dose requirements of the elderly.

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Pethidine compared with meptazinol during labour

A prospective randomised double-blind study in 1100 patients

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Summary

A randomised double-blind comparison of pethidine and meptazinol used as analgesics in labour was carried out in 1100 consecutive women who would normally have received intramuscular pethidine. Pain assessments at 30-minute intervals were made independently by patients and midwives. Maternal and neonatal side effects were noted. The babies' requirements for resuscitation and weight changes in the first 5 days were studied. There was no difference in the analgesia provided by the two drugs; the pattern of side effects was similar, but the incidence of vomiting was greater following meptazinol administration. The babies in the two groups were similar with respect to resuscitation received, weight gains or losses and the incidence of clinical neonatal jaundice. The most striking findings were the poor quality of pain relief experienced by both groups following parenteral analgesics and the high incidence of side effects.

Key words

Pain; obstetric.
Analgesics, narcotic; pethidine, meptazinol.

The most commonly prescribed parenteral analgesic during labour in the United Kingdom is pethidine.[1] It has several disadvantages, namely poor analgesia,[2] neonatal depression,[3,4] a high incidence of vomiting and other maternal side effects. It is also time consuming to administer since it is a controlled drug. It has been suggested that meptazinol has advantages for this use because it has more rapid onset of analgesia with fewer side effects. There is also the possibility of advantages to the baby, because meptazinol has a much shorter half-life in the neonate than pethidine. This may predispose to earlier and better feeding.[5]

This study was set up to compare, in a prospective, randomised, double-blind fashion, the effects of pethidine and meptazinol in 1100 consecutive women in labour and their babies.

Method

This trial, which had ethics committee approval, was conducted in Rutherglen Maternity Hospital which has approximately 3000 deliveries a year. The study comprised 1100 consecutive patients who would normally have received intramuscular pethidine. The only exceptions were 12 patients inadvertently prescribed pethidine instead of

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'trial drug' (see below) and one patient who did not wish to take part in the trial. The normal policy of analgesia in labour was not altered during the course of the trial. Other forms of analgesia, such as epidural or inhalational, were administered as required. When pethidine was to be the drug used, 'trial drug' was substituted. This consisted of randomly numbered batches of pethidine or meptazinol, each containing three 100-mg ampoules to ensure that in subsequent doses the patient received the same drug. Informed consent was obtained from the patients who were also told they would be offered additional analgesia if required. Mothers weighing more than 70 kg were given 150 mg, while the remainder received 100 mg. The trial drug ampoules were coded and the information kept available to the medical staff if required and to the hospital pharmacist. The code did not require to be broken before the end of the trial.

The patient and attending midwife independently completed a 100-mm visual analogue scale assessing the mothers' pain at the time of administration of the drug and at 30, 60, 90 and 120 minutes thereafter.[6,7] The maternal blood pressure was also recorded at these times. The time in relation to the injection, of any nausea or vomiting, pre-injection nausea or vomiting, supplementary analgesia, duration of labour and the method of delivery were also recorded. The patients' age, weight, parity, gestation and pre-labour blood pressure were available from the case sheet.

The mothers were interviewed between the 3rd and 5th postpartum days and the incidence of euphoria, dysphoria, sleepiness and dizziness was noted. The mothers were also asked to complete the following four-point verbal rating scale, to record their opinion of the analgesia provided by the 'trial drug': excellent, good, poor but just able to cope, or no effect and required additional analgesia.

The following information about the babies was collated: incidence of fetal distress, incidence of naloxone administration, resuscitation received, Apgar scores at 1,5 and 8 minutes, birth weight, type of feeding (breast or bottle), weight gains or losses in the first 5 days, incidence of clinically significant jaundice in the neonatal period, serum bilirubin level in affected babies.

Statistical methods

The data were analysed by the Statistical Package for the Social Sciences (SPSSX) on the University of Glasgow ICL 2988 mainframe computer. The distributions of categorical and grouped continuous variables in the pethidine and meptazinol groups were compared by the Chi squared test. The mean values of continuous variables were compared by the two sample *t*-test.

Results

The results of 65 mothers were eliminated from analysis for various clerical reasons, such as lost case sheets, or prescribed mixed drugs (pethidine and meptazinol given to the same patient at different times). Of the 1035 cases analysed, 522 received pethidine and 513 meptazinol. The groups were comparable with regard to age, maternal weight, parity and gestation (Table 1).

Pattern of treatment received by each group

Second injections of the analgesics were given to 71 mothers (13.6%) in the pethidine group and 55 (10.9%) who received meptazinol. Of these, 23 pethidine mothers and 13 meptazinol mothers received the second injection within 2 hours of the first. The mean interval between injections was 2.54 hours (SD 0.81) for those given pethidine and 2.72 hours (SD 0.85) for those given meptazinol, which was not significantly different. The epidural rate in the two groups was the same at 30%.

Table 1. Details of mothers.

	Age, years (SEM)	Weight, kg (SEM)	Primipara	Multipara	Gestation <38 weeks	 38–42 weeks	 >42 weeks
Pethidine group	26.2	70.5	230	292	41	471	9
	(4.4)	(10.2)	(44.1%)	(55.9%)	(7.8%)	(90.4%)	(1.7%)
Meptazinol group	26.0	69.5	224	289	50	457	5
	(4.6)	(9.5)	(43.7%)	(56.3%)	(9.8%)	(89.3%)	(1.0%)

Assessment of analgesia

During labour. The results of the patients' pain scores are shown in Table 2. Only 339 patients had pain scores measured for the full 120 minutes; there were 218 primigravida and 121 multipara in this group. No, or only one, pain score was recorded for 239 patients, so these cannot be included in an analysis of changes in pain score. This was due to the patient delivering less than 60 minutes from the injection (105 pethidine, 118 meptazinol) and to 16 patients with records unavailable due to pressure on nursing staff or loss of the pain scores.

The remaining 457 patients had pain scores recorded for less than 120 minutes. This comprised 122 pethidine and 125 meptazinol mothers delivered more than 60 minutes but less than 120 minutes after the injection, and 47 pethidine and 38 meptazinol mothers delivered just after 120 minutes, making it difficult to obtain a 120-minute score. The remainder comprise the mothers who received second injections less than 120 minutes after the first and a further 78 mothers (40 pethidine, 38 meptazinol) who received an epidural within 120 minutes of the injection.

The findings in each of these groups (all patients, complete, primiparous and multiparous) are included, as it might be argued that there was loading of primipara in the 'complete' group. Overall, there was no statistically significant difference between the pethidine and the meptazinol mothers. Midwives' scores followed the same pattern as that of the patients, but were consistently 6–10 points less.

Postpartum. The answers to the postpartum verbal rating scale from the 407 mothers given pethidine and the 394 given meptazinol are shown in Table 3. There was significant difference neither between the two groups, nor in the number who would wish to receive the same drug in a subsequent labour.

Patterns of side effects in the two groups

Euphoria was reported by 14% of mothers in each group and dysphoria by 11%. Significantly more mothers given pethidine (202) compared to meptazinol (147) experienced sleepiness ($p < 0.001$), while 95 of the former and 125 of the latter reported dizziness ($p < 0.01$). Excluding 15 pethidine and 15 meptazinol mothers who experienced nausea and vomiting pre-injection, the

Table 2. Patients' pain scores as recorded on a 100-mm visual analogue scale: mean (SD).

Patient group:	All patients		Complete*		Primiparous		Multiparous	
Treatment:	Pethidine	Meptazinol	Pethidine	Meptazinol	Pethidine	Meptazinol	Pethidine	Meptazinol
At injection	74.7 (17.4)	73.8 (18.9)	73.4 (17.8)	72.8 (18.9)	75.5 (16.6)	70.9 (18.9)	73.9 (18.1)	76.3 (18.6)
After 30 minutes	67.0 (21.8)	64.9 (21.5)	59.4 (22.6)	58.2 (20.8)	63.6 (22.5)	59.9 (20.1)	70.4 (20.7)	69.0 (21.8)
After 60 minutes	67.4 (23.7)	68.5 (22.5)	60.7 (23.9)	61.8 (22.1)	64.9 (23.6)	63.8 (23.0)	70.4 (23.6)	73.6 (20.7)
After 90 minutes	73.6 (20.7)	70.6 (22.9)	70.2 (20.6)	68.6 (23.4)	70.3 (22.0)	68.6 (23.0)	78.8 (17.2)	73.6 (22.5)
After 120 minutes	77.9 (20.7)	78.3 (21.5)	78.1 (20.6)	78.3 (21.6)	77.6 (19.9)	76.6 (22.3)	78.6 (22.3)	81.5 (19.8)

*Recordings complete for 120 minutes after injection.

Table 3. Results of postpartum assessment of analgesia.

Verbal rating:	Excellent	Good	Poor, just able to cope	No effect, needed something else	Would have same drug again in future labour
Pethidine group ($n=407$)	10 (2.5%)	137 (33.7%)	102 (25.1%)	158 (38.8%)	187 (45.2%)
Meptazinol group ($n=394$)	7 (1.8%)	132 (33.5%)	119 (30.2%)	136 (34.5%)	190 (48.0%)

Table 4. Incidence of fetal distress.

	Pethidine ($n=522$)	Meptazinol ($n=513$)
Stage 1		
Early deceleration (%)	30.5	27.9
Late deceleration (%)	6.1	8.6
Fetal tachycardia (%)	4.4	2.1
Stage 2		
Early deceleration (%)	50.8	49.5
Late deceleration (%)	16.1	16.2
Fetal tachycardia (%)	2.5	2.3
One or more of the above	67.6	67.1

Table 5. Method of delivery.

Method of delivery:	Spontaneous vaginal (%)	Rotation forceps (%)	Other forceps (%)	Caesarean section (%)
Pethidine group ($n=521$)	74.7	2.3	18.3	4.8
Meptazinol group ($n=512$)	74.6	2.9	16.1	6.4

incidence of nausea in those who received pethidine was 33% (169 mothers) compared to 38% (189 mothers) given meptazinol ($p<0.01$). The corresponding figures for vomiting were 28% (141) and 37% (184), respectively, which again reached statistical significance. The time pattern was also different in the two groups; nausea and vomiting occurred significantly later after meptazinol ($p<0.01$).

Effect of drug on maternal blood pressure

Neither pethidine nor meptazinol appeared to exert any significant effect on maternal blood pressure. The pre-labour recordings were similar in the two groups.

Neonatal results

There was no difference between the two groups with regard to neonatal outcome. The mean weight of the pethidine babies was 3.4 kg (range 1.78–4.82) and of the meptazinol babies 3.4 kg (range 1.40–5.52). The incidence of fetal distress during labour is shown in Table 4 and the method of delivery in Table 5. There was no difference between the two groups with regard to the various modes of delivery. Twenty percent of babies were delivered within 60 minutes of analgesic administration and 40% more than 180 minutes after receiving the narcotic.

Slightly more babies whose mothers received pethidine were given naloxone, but the difference was not statistically significant (Table 6). However, when analysing the use of naloxone in relation to the time between analgesic administration and delivery, it was found that significantly more 'pethidine babies' (53) who were delivered more than 180 minutes following the narcotic required naloxone compared to the 'meptazinol babies' (31), $p < 0.05$. The outcome for babies of less than 36 weeks gestation is also shown in Table 6, but the numbers are too small for any definite conclusions to be drawn.

Table 6. Neonatal information.

	Neonatal information		<36 weeks	
	Pethidine group ($n=496$)	Meptazinol group ($n=479$)	Pethidine group ($n=8$)	Meptazinol group ($n=15$)
Naloxone required	231 (46.6%)	198 (41.3%)	5 (62.5%)	9 (60.0%)
Injection–delivery interval				
<60 minutes	50 (47.6%)	53 (44.9%)		
60–180 minutes	133 (63.9%)	122 (60.7%)		
>180 minutes	53 $p < 0.05$ (25.5%)	31 (16.0%)		
No resuscitation	55 (11.2%)	50 (10.4%)	0	2 (13.4%)
Oropharyngeal suction, O_2 via mask	384 (77.4%)	375 (78.3%)	6 (75%)	8 (53.3%)
Tracheal intubation	57 (11.5%)	54 (11.3%)	2 (25.5%)	5 (33.3%)
Apgar scores, mean (range)				
1 minute	7.5 (2–10)	7.5 (1–9)	6.8 (2–10)	6.7 (2–9)
5 minutes	9.1 (4–10)	9.2 (4–10)	9.0 (7–10)	8.5 (7–10)
8 minutes	9.8 (7–10)	9.8 (7–10)	9.9 (9–10)	9.4 (7–10)

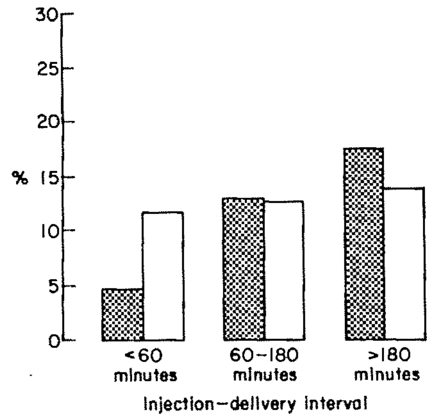

Fig. 1. Percentage of babies in each injection–delivery interval requiring tracheal intubation. ▦, Pethidine; ☐, meptazinol.

Table 6 shows the number of babies requiring resuscitation, either by oropharyngeal suction and oxygen administration by facemask or tracheal intubation. There was no difference between the groups. Figure 1 shows the proportion of babies requiring tracheal intubation related to the narcotic administration–delivery interval. Whereas the proportion of babies requiring tracheal intubation was similar in both groups, the number who required intubation in the pethidine group increased as the injection–delivery interval increased. In contrast, the number remained reasonably constant at each injection–delivery interval in the meptazinol group.

There was no difference between the babies with respect to the Apgar scores at 1, 5 and 8 minutes (Table 6). However, the Apgar scores in the pethidine group at 5 minutes showed a significant decrease with increasing injection–delivery interval. The weight changes of the babies at 3 and 5 days are shown in Table 7; there was no difference between the groups. There was no significant difference in the incidence of jaundice postdelivery, nor in the use of phototherapy.

Discussion

The place of pethidine as an analgesic in labour is established and hallowed by time, although its acceptability and efficacy are much less than satisfactory.[2] The search for an alternative drug with more potent analgesia and fewer maternal and neonatal side effects continues. It has been suggested that meptazinol may have advantages for the mother and neonate.[5] This study does not confirm that opinion. Using the midwives as objective assessors, there was good correlation between the subjective and objective pain scores. The analgesia obtained was shown to be greatest at 30 and 60 minutes after the injection when either drug was used, but neither decreased to a

Table 7. Weight gains or losses of babies.

		Pethidine group (breast)	Meptazinol group (breast)	Pethidine group (bottle)	Meptazinol group (bottle)
Day 3	*n*	194	214	318	296
	Zero or +	1.5%	2.8%	5.6%	7.4%
	1–5% loss	38.1%	45.8%	73.9%	67.9%
	6–10% loss	56.2%	49.5%	19.8%	23.6%
	10% loss	4.1%	1.9%	0.6%	1.0%
Day 5	*n*	185	195	296	274
	Zero or +	8.1%	12.8%	13.5%	14.2%
	1–5% loss	56.2%	58.5%	70.6%	67.9%
	6–10% loss	34.0%	28.2%	15.2%	17.5%
	10% loss	1.6%	0.5%	0.7%	0.4%

satisfactory level. This does not agree with the findings of Nicholas and Robson,[5] who found that during the first hour after injection there was a significantly greater decrease in pain scores with meptazinol. This may be explained by the parity of the women in their trial, which is not specified. We have found that primigravid women had a substantially greater decrease in pain scores after both drugs. Thus, groups of women not similar in respect of parity may invalidate the comparisons of pain scores. We have also shown that although the mean changes in pain scores were small, there were wide individual variations as indicated by the large standard deviation of the mean scores. (The midwives' scores were consistently 6–10 points less than those of the patients.)

The time interval between the first and second injections of either pethidine or meptazinol was similar and had a fairly wide variation, although 50% of those receiving a second injection did so within 2.5 hours. This timing coincides with the changes in pain scores which had returned to, or were greater than, their original level by 120 minutes. Both groups had a subsequent epidural rate of 30%. The similarity between the groups is despite our acceptance of the equipotency of pethidine and meptazinol at 100 mg and 150 mg doses in labour, which has been challenged.[8,9] It is also in contrast to the small trial of Vickers and Rosen.[10]

The overall epidural rate increased from 35% to 40% during the period of the trial and reduced when the trial was complete. Speculation as to the reason perhaps points to a lack of confidence in the 'trial drug' by the staff, or an increased awareness of patient pain with its assessment every 30 minutes.

Meptazinol produced no fewer side effects than pethidine and, apart from the differences in sleepiness and dizziness rates in the two groups, the same spread of side effects was seen. Perhaps more surprising than the similarity between the groups is the fact that 66% of mothers in both groups experienced at least one side effect and this can certainly be considered to be less than acceptable. The incidence of nausea and vomiting before analgesia in this group of patients was 3% and, accepting that there would be an increased incidence as labour progressed, the vomiting rate in both groups after treatment was fairly high, but significantly greater in the meptazinol group. Despite the numerical significance of this difference, it may be considered that in a clinical situation there is no difference as neither vomiting rate is acceptable.

The time distribution of the vomiting is hard to explain. It might be expected to have been related to the blood levels of the drug or to the analgesia produced. We have shown that the onset of analgesia and the drop in pain scores is similar in both groups. Thus, the earlier onset of vomiting following pethidine and the later onset after meptazinol are not related to the analgesia produced and the assumption is that these observations are not therefore related to the blood levels of the drugs. We would conclude that meptazinol, in an equi-analgesic dose, gives a greater incidence of vomiting than pethidine, often with a later time of onset.

The theoretical advantages of meptazinol over pethidine to the neonate are due to the difference in the metabolism of the two drugs. Meptazinol undergoes glucuronidation in the liver, whilst pethidine undergoes ester hydrolysis, N-demethylation and N-oxidation reactions which are known to be much reduced in the neonate. These routes are reflected in a neonatal half-life for pethidine of 22.7 hours compared with 3 hours in the adult and for meptazinol 3.4 hours compared with 2.2 hours in the adult.[11,12] The many deleterious effects of pethidine on the neonate[3,4] may therefore be expected to be less and of shorter duration when the mother receives meptazinol.

The injection–delivery interval with the worst outcome for the neonate, as reflected in the requirements for resuscitation, is 60–180 minutes for the pethidine group. This agrees with the findings of Morrison *et al.*,[13] and also Schneider and Moya[4] who showed that the neonatal depression seen in the babies of pethidine-treated mothers is partly due to the metabolites of pethidine, rather than solely to the unmetabolised drug. In the meptazinol group babies, although the percentage requiring resuscitation is the same as in the pethidine group, the number of meptazinol group babies in each injection–delivery interval requiring each form of resuscitation is similar. We conclude that the injection–delivery interval is not an important factor in the neonatal depression seen in the babies of meptazinol-treated mothers. The similarity between the groups in the total number of babies requiring resuscitation is unexpected in that the maternal/fetal plasma concentration is more for pethidine, 1.00 and 0.25 (mean, SEM), than for meptazinol, 0.57 and 0.05 (mean, SEM).[11,14,15] Thus, a larger percentage of depressed pethidine babies might have been expected. The Apgar scores of the two groups of babies correlate with the above conclusions.

The fact that 40% of babies were given naloxone requires some comment. There was no difference in the incidence of administration between the groups and the high usage reflects the local practice at the time of midwives being allowed to administer naloxone 0.02 mg on their own account, often for such reasons as 'late sedation' or 'sleepiness'. Of the babies given naloxone, 41% had an Apgar score of 8 or more at 1 minute and a further 25% a score of 7. At 5 minutes, 91% of these babies had an Apgar score of 9 or more. Thus, it is obvious that naloxone administration was inappropriate in most of the babies.

The weight gains or losses of babies may well be an indication of their ability to suck. It has been shown that pethidine depresses this ability.[16] In breast-fed babies, any difference between the groups would be expected to be more obvious on the 5th than on the 3rd day, as by then lactation will be well established. The quantity of milk would be greatest in mothers with babies who sucked well. There was a trend in the breast-fed group for more meptazinol than pethidine babies to be in the 'gain' or 'up to 5% loss' groups and more pethidine group than meptazinol group babies to be in the 'up to 10% loss' and 'greater than 10% loss' groups. This was both on the 3rd and 5th days, but did not reach statistical significance.

The injection–delivery interval did not affect the weight changes in the breast-fed or bottle-fed babies in either group on day 3 or the meptazinol babies on day 5. However, in the breast-fed pethidine babies on day 5 there was a trend for more babies in the 60–180-minute injection–delivery interval to be in the gain or < 5% weight loss group and for more babies in the greater than 180-minute injection–delivery interval to be in the > 5% weight loss group, but again this did not reach statistical significance.

The incidence of clinical neonatal jaundice, while similar in the two groups, did show a difference in the severity of the jaundice when the requirements for phototherapy were compared. Physiological jaundice is also related to the ability to suck,[17] but neither the differences in weight gains and losses nor the differences in phototherapy requirements reached statistical significance.

In conclusion, meptazinol does not appear to have any clinical advantages over pethidine as a routine narcotic analgesic in labour. It does not produce a more rapid onset, nor a better quality of analgesia in the mother and it produces a significantly higher incidence of vomiting. Theoretically the neonate should benefit from the shorter half-life of meptazinol as compared with pethidine, but in our study of 1100 deliveries this was not found to be the case with reference to the resuscitation required, the Apgar scores or the babies' ability to suck, as reflected in the weight gains and losses in the first 5 days of life, or the incidence of neonatal jaundice. This study does show clearly, however, how far we fall short of providing satisfactory analgesia with intramuscular pethidine for delivery.

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The prevention of renal impairment in patients undergoing orthotopic liver grafting by infusion of low dose dopamine

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Summary

Administration of low dose dopamine (2.0 µg/kg/minute) begun before surgery in patients undergoing liver transplantation decreases the incidence of postoperative renal impairment. Thirty-four consecutive patients in the Cambridge/King's College Hospital liver transplantation series were studied. Nineteen patients (21 transplant operations) received prophylactic low dose dopamine throughout the operative and early postoperative period, while 15 patients (15 transplant operations) received dopamine only when clinically indicated for incipient renal failure or as an inotropic agent. In the prophylactic dopamine group, only two transplant operations (9.5%) were complicated by renal impairment, whereas in the other group, 10 patients (67%) developed renal impairment ($p = 0.001$); of these, four developed acute renal failure (27%). Comparison of seven pairs of patients, matched for age, sex, diagnosis, operative blood loss and operative hypotension *(one group receiving dopamine, the other not), revealed a significantly higher urine output in the first 24 hours and creatinine clearance 24–48 hours after surgery ($p < 0.05$) in those treated prophylactically. In view of these findings, we would recommend that consideration be given to the prophylactic use of dopamine in patients undergoing orthotopic liver transplantation.*

Key words

Surgery; liver transplantation.
Complications; acute renal failure.
Inotropes; dopamine.

Patients undergoing orthotopic liver transplantation may develop impaired renal function during the operation or in the early postoperative period and occasionally require haemodialysis. This may be due to several interacting factors, of which major blood loss occurring in the peri-operative period appears to be the most important. Haemorrhage results in sympathetic vasoconstriction, which mainly affects the afferent glomerular arterioles, with a fall in renal blood flow and consequent renal ischaemia. Septicaemia and the use of potentially nephrotoxic drugs, such as cephalosporins or aminoglycoside antibiotics (especially in combination with loop diuretics) and the immunosuppressant cyclosporin A,[1] may also be causally implicated.

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Dopaminergic stimulation may reverse this sympathetic vasoconstriction and so help prevent the renal ischaemia.[2] Dopamine hydrochloride in the dose range 2–5 μg/kg/minute is a potent renal dopaminergic agonist and is commonly used to treat patients with incipient renal failure,[3] so its administration prior to and during major haemorrhage would seem worthy of consideration. In this report we describe our experience with the prophylactic use of an infusion of dopamine 2 μg/kg/minute, in patients having orthotopic liver transplant operations; the drug was started prior to surgery and continued into the postoperative period.

Patients and methods

In an 18-month period, 34 patients were treated by orthotopic liver transplantation; three of these patients required retransplantation within the same period. One patient with primary hyperoxaluria and renal failure, who had a combined liver and repeat kidney graft operation, was treated electively with haemodialysis both pre- and postoperatively and was therefore excluded from further analysis. All patients received 10% mannitol 1 g/kg during the procedure and all were given benzylpenicillin, cefoxitin and tobramycin during the operation and for 48 hours postoperatively. For immunosuppression, prednisolone and azathioprine were used; oral cyclosporin A was introduced in the second postoperative week in order to avoid the renal and pulmonary toxicity associated with intravenous administration immediately after operation.[4] Details of the anaesthetic and surgical techniques have been described previously.[5,6]

Nineteen patients (21 transplant operations) received a low dose dopamine infusion of 2 μg/kg/minute, begun with the induction of anaesthesia and continued for 48 hours postoperatively (group 1), while 15 patients (15 transplant operations) did not (group 2). A patient who required retransplantation for chronic rejection 6 months after her first transplant operation was treated with dopamine prophylactically only during her second operation and so was included in both groups. During this period there was no formal policy about the use of prophylactic dopamine; the decision whether or not to use it was made by the consultant anaesthetist involved.

The peri-operative details of these two groups were compared, as was the incidence of renal impairment defined as oliguria (less than 0.5 ml/kg/hour urine output) in the presence of an adequate central venous or pulmonary capillary wedge pressure and a urine/plasma osmolality ratio of 1.1:1.0 or less,[7] or acute renal failure requiring haemodialysis. In accordance with conventional management, a dopaminergic dose of dopamine was begun immediately a patient in group 2 developed renal impairment.

To determine whether bypass support, which diminishes venous congestion during the anhepatic phase of the operation, had a beneficial effect on renal function, the results for the five patients in the dopamine-treated group in whom veno-arterial bypass[8] was used and for the six patients in the untreated group in whom veno-venous bypass[9] was used, were compared with the remaining patients in their respective groups. Similarly, as we have previously shown that younger patients have better survival rates than older patients after liver transplantation,[10] the renal function of those aged 21 years or under was compared with that of the older patients within each treatment group.

Six pairs of patients were matched for age, sex and diagnosis and as closely as possible for pre-operative weight, plasma creatinine concentration and intra-operative blood loss and degree of hypotension. One of each pair was receiving low dose dopamine. The one patient who required retransplantation for chronic rejection 6 months after her first operation was included as the seventh 'pair'.

Statistical analysis was by the unpaired Student's *t*-test, the Mann–Whitney U test and Fisher's test of exact probability where appropriate.

Results

The two groups of patients were similar with respect to age, sex, diagnoses, pre-operative plasma creatinine and operative blood loss (Table 1). In group 1, only two (9.5%) of the 21 transplant operations were complicated by acute renal failure. A 13-year-old female who had compromised renal function before operation, with a plasma creatinine concentration of 150 μmol/litre, required haemofiltration from 6 days postoperatively and haemodialysis from 13 days, while a second patient (a 46-year-old male) required haemofiltration from 2 days and haemodialysis from 10 days postoperatively.

Table 1. Renal function in 34 consecutive patients undergoing orthotopic liver transplantation from 1 May 1983–31 October 1984.

	Prophylactic dopamine*	No prophylactic dopamine*
Number of transplant operations	21	15
Number of patients	19 (7M : 12F)	15 (6M : 9F)
Age, years		
Mean (SEM)	29.7 (4.3)	34.5 (4.3)
Range	2–58	2–57
Diagnosis		
Biliary atresia	3	1
Inborn errors of metabolism	1	1
End stage liver disease:		
Primary biliary cirrhosis	5	4
Chronic active hepatitis	3	2
Others	6	3
Malignant liver disease	1	4
Operative blood loss, litres		
Mean (SEM)	9.5 (2.9)	7.3 (1.7)
Range	0.3–54.0	1.0–22.2
Pre-operative plasma creatinine (concentration, μmol/litre		
Mean (SEM)	75.9 (6.6)	79.0 (4.8)
Range	45–150	46–102
Outcome		
Renal impairment at 2 weeks	2/21 (9.5%)	10/15 (67%)
Acute renal failure at 2 weeks	2/21 (9.5%)	4/15 (27%)

*One patient who was treated with prophylactic dopamine during her retransplant operation, but not for her initial operation, is included in both groups.

In contrast, of the 15 patients (15 transplant operations) in group 2 not electively treated with dopamine, 10 patients (67%) developed renal impairment, a statistically significant difference ($p = 0.001$). In three cases this occurred during the operation; one patient subsequently required haemodialysis from the sixth postoperative day; six patients required dopamine within the first 24 hours after operation and three of these required haemodialysis within the first postoperative week, while the remaining patient started dopamine during the second postoperative day.

The incidence of renal impairment in patients in whom bypass support was used was not significantly different from that in the remaining patients in their respective groups. Likewise, no difference was found when the results within the two treatment groups were analysed with respect to age.

When the results for the paired patients were compared, it was found that the volume of urine produced on the first postoperative day and the creatinine clearance in the period 24–48 hours after operation were significantly better in those patients who received prophylactic dopamine than in those who did not ($p < 0.05$; Table 2). There was no significant difference between the pre-operative renal function, the operative blood loss or the lowest recorded systolic blood pressure in the two groups.

Discussion

This study suggests that the prophylactic administration of dopamine, begun prior to the onset of surgery, decreases the incidence of renal impairment postoperatively in patients undergoing liver transplantation. Furthermore, although the numbers are small, renal failure requiring haemodialysis or continuous arteriovenous haemofiltration occurred less often and much later when prophylactic dopamine was given. The problems associated with results obtained from a retrospective study are well known; however, the dramatic fall in morbidity and mortality associated with the continuing use of prophylactic dopamine has been maintained since the end of the study period and we could not now ethically consider a prospective study in these patients. The inclusion in this study of a

Table 2. Renal function in paired patients. The first patient in each pair was treated with prophylactic low dose dopamine.

	Pairs														Statistics
	1		2		3		4		5		6		7		
Patient number	166	164	165	145*†	159	150†	157	124	170	147	162	117†	148b	148a†	—
Age (years)/sex	46F	46F	50F	48F	2M	2M	17F	17F	13F	10F	52M	47M	11F		—
Diagnosis	PBC		PBC		BA		CAH		Hepatoma		AC		R	SAHN	—
Weight (kg)	50	44	58	60	13.8	12.0	52	50	35	36	80	81	40	36	NS
Pre-op. plasma creatinine (μmol/litre)	91	64	70	69	—	—	61	119	49	58	97	108	46	107	NS
Blood loss (litres)	3.8	6.0	7.9	5.0	0.7	2.3	7.4	4.1	0.3	3.9	16.6	7.9	11.3	3.9	NS
Lowest systolic BP (mmHg)	80	90	100	80–85	90	75–80	95	85	70	80	65–70	90	80	90	NS
Urine output (ml)															
0–24 hours	1420	1700	2240	490	720	181	1010	550	1530	940	2040	630	1190	370	p<0.05
24–48 hours	2710	1730	2020	170	440	550	1850	2400	1880	780	4480	150	1260	740	NS
Creatinine clearance (ml/minute)															
0–24 hours	74	50	70	9	15	61	51	35	108	90	74	13	30	8	NS
24–48 hours	64	39	77	1	11	12	103	44	94	52	129	1	23	10	p<0.05

* Required haemodialysis 72 hours postoperatively.

† Required dopamine 0–24 hours postoperatively.

PBC = Primary biliary cirrhosis; BA = biliary atresia; CAH = chronic active hepatitis; AC = alcoholic cirrhosis; R = rejection; SAHN = subacute hepatic necrosis.

matched pair group overcomes some of the difficulties associated with a retrospective study and tends to reinforce the conclusions drawn from the findings of the larger unmatched group.

The use of bypass to decompress the portal circulation and to maintain the systemic blood pressure and renal perfusion, made no difference to the frequency with which renal impairment occurred, contrary to the report by Starzl *et al.*[9] Also, the patients' age did not appear to alter the incidence of renal impairment, in contrast to its effect on overall mortality, in which the older patients did less well.[10]

The introduction of cyclosporin A has improved immunosuppression following organ transplantation.[11] However, its use in the early postoperative period following liver transplantation is limited by its nephrotoxicity. This necessitates the use of azathioprine, with the hazards of marrow suppression, and of high dose steroid therapy, with the disadvantages of an increased risk of, and difficulty in, the diagnosis of infection and adverse effects on carbohydrate metabolism which make parenteral nutrition difficult. Preliminary studies have shown that intravenous cyclosporin A results in a marked decrease in renal blood flow (P.R. Powell-Jackson, personal communication) and it may be that simultaneous low dose dopamine infusion will prevent, or at least lessen, this decrease and thus reduce toxicity, thereby allowing earlier postoperative use of this agent. Furthermore, if severe cyclosporin A nephrotoxicity can be avoided initially, the chances of long-term kidney damage may be reduced. Recently, a patient who was treated immediately after operation with cyclosporin A, given through a central venous line, developed severe pulmonary toxicity, but the anticipated nephrotoxicity did not develop, possibly due to the continued administration of dopamine.

Care is necessary in the extrapolation of results from liver transplant patients to others, but it seems appropriate to treat patients undergoing surgical procedures in which major haemorrhage occurs (such as ruptured abdominal aneurysms or following polytrauma) and who may be at risk of developing acute renal failure, with a dopamine infusion at a rate of 2 μg/kg/minute throughout the operative and immediate postoperative periods.

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Quality of axillary brachial plexus block

Comparison of success rate using perivascular and nerve stimulator techniques

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P. H. ROSENBERG

Summary

A perivascular catheter technique (PVT) and a nerve stimulator technique (NST) for axillary brachial plexus block were compared in terms of quality: complete, incomplete or failed blocks. In a randomised series, 30 PVT blocks and 30 NST blocks were performed by three staff anaesthetists. In the NST group, surgical anaesthesia was always achieved, whereas in the PVT group, four blocks required supplementation with general anaesthesia. In both groups eight patients needed supplementation with additional conduction blocks of 1–3 peripheral nerves. It is concluded that a nerve stimulator technique may increase the success rate of axillary brachial plexus block to some extent.

Key words

Regional techniques; brachial plexus block.

It is believed that the use of a nerve stimulator to identify a nerve plexus provides an increase in both safety and success rate.[1–5] The perivascular technique for axillary brachial blocks,[6] as modified by Selander,[7] was used here for some 5 years, after which the method was replaced by the nerve stimulator technique. In a retrospective review of our results, surprisingly no difference was found between the quality of 200 blocks performed with the two different techniques. It was therefore decided to compare these two techniques under controlled conditions in a prospective study, in order to establish their efficacy.

Patients and methods

Sixty axillary brachial blocks for elective surgery were performed by three senior anaesthetists experienced in regional anaesthesia. In random order, half of the patients received an axillary brachial plexus block *via* the perivascular technique with a catheter as described by Selander[7] (PVT group). In the remaining patients, blocks were performed with the aid of a nerve stimulator in order to identify the axillary nerve plexus (NST group). No effort was made to distinguish between the three main nerve trunks. A Neurostim LA nerve stimulator, type 218 (Hugo Sachs Elektronik KG, FRG)[8] and regional anaesthesia needles (50 mm, 22-G) with an electro-neurostimulation port (Vygon, France) were used.

Bupivacaine 0.5%, approximately 3 mg/kg, was given in a volume of 40 ml to patients weighing 50–70 kg, 50 ml for those 71–90 kg in weight and 60 ml for patients weighing over 90 kg. The volume was supplemented with physiological saline when needed.

Analgesia in defined areas of the ulnar, radial, median and musculocutaneous nerves was examined by pin-prick at 10-minute intervals, as

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was the motor blockade of the hand and elbow. The sensory block was classified as: complete, incomplete, failed. The classification complete, also included those patients who had been given a small dose of sedative (diazepam $\leqslant 5$ mg or lorazepam $\leqslant 2$ mg) or analgesic (fentanyl $\leqslant 0.1$ mg) during the course of surgery. After 30 minutes, if analgesia was incomplete, the blocks were supplemented with additional conduction blocks of the ulnar, radial, median or musculocutaneous nerves. If the block then still failed to provide satisfactory surgical analgesia, general anaesthesia was administered.

The statistical significance of the differences between the means was calculated using Student's *t*-test and between the frequencies using the Chi squared test with Yates' correction for small numbers.

Results

The patients in this series were comparable except for age, where the PVT patients were older ($p < 0.05$). The ratios of complete blocks in the PVT and NST groups were 18/30 and 22/30, respectively. In each group, eight patients required an additional conduction block of one to three nerves. In the PVT group, four blocks failed to give surgical anaesthesia; in the NST group, none of the blocks failed (Table 1). However, when all the blocks were combined, the difference in the success of the blocks (three grades) between the two groups was not statistically significant.

Table 1. Characteristics of the patients and success rate of two techniques of axillary brachial plexus block.

	Perivascular catheter technique	Nerve stimulator technique
Number of patients	30	30
Sex M/F	21/9	23/7
Age, years (SD)	46 (15)*	37 (12)
Weight, kg (SD)	80 (17)	76 (15)
Range	48–118	54–113
Height, cm (SD)	172 (8)	174 (9)
Range	154–187	157–189
Success rate		
Total	30	30
Complete block	18	22
Incomplete block	8	8
Failed block	4	—

* $p < 0.05$.

One of the PVT patients became unconscious and convulsed 2 minutes after the injection of bupivacaine (205 mg) was given. The venous bupivacaine plasma levels 5 and 15 minutes later were 7.0 μg/ml and 4.8 μg/ml, respectively. The patient received 25 mg diazepam intravenously, the trachea was intubated and the lungs were ventilated with 100% oxygen. Within 30 minutes he had regained consciousness and could be extubated. No cardiac dysrhythmias or haemodynamic depression were evident when the convulsions were under control, the ECG could be clearly seen and the blood pressure measured. There was no analgesia in the hand or arm.

Discussion

When a perivascular axillary brachial block technique has been used by experienced anaesthetists, the incidence of inadequate block for surgery is 5–6%.[7,9,10] Notwithstanding this, reports showing no failures in a series of axillary plexus blocks, as performed by a single anaesthetist, have also appeared.[11] In the present series, there was a failure rate of 6.7% when the blocks were carried out by three anaesthetists; all four failures occurred in the PVT group. In the NST group, 22 blocks were complete; eight incomplete blocks required supplementation by performing additional conduction blocks of the peripheral nerves in order to give surgical anaesthesia. Statistically, there was no difference between the success of the axillary blocks performed with the two techniques.

An explanation for the comparatively high number of incomplete blocks in the NST group may be that it was not intended to stimulate a specific axillary nerve trunk, but rather to establish the best response in the hand, obtained using the lowest current as an index. Yasuda *et al.*[5] noted that the supraclavicular plexus block was more successful when the stimulation was sensed in the index, middle and ring fingers rather than in the thumb or fifth finger.

One of the principal advantages with either PVT or NST for axillary blocks is that in neither technique is it necessary to aim for paraesthesia. Furthermore, the risk of mechanical nerve injury is low.[4,7] In both methods, a complete or successful block also depends on keeping the needle or the catheter tip within the neurovascular sheath throughout the injection of the local anaesthetic solution. Re-identification as well as

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identification of the other main nerve trunks by electrical stimulation is impaired after injection with only a small volume of a local anaesthetic solution into the vicinity of the nerve.[4]

In addition to the case of severe intoxication in the present series, three accidental intravenous injections occurred; these produced varying reversible CNS symptoms when NST was used for axillary or interscalene approaches to the brachial plexus. In retrospect, it seems that the efforts to aspirate were insufficient and blood could not be detected because the connecting tubing of the neurostimulator–needle–injecting set (Vygon) is opaque and 35 cm long. Otherwise, with the doses of bupivacaine used in the present study, toxic blood concentrations were not to be expected.[12]

In conclusion, when 60 axillary brachial blocks were randomly performed by three experienced anaesthetists using either technique, four failures (6.7%) were encountered; all of these were associated with the perivascular technique. However, the general success rate, graded as complete, incomplete or failure, was not statistically different between the two techniques.

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Anaesthesia, 1987, Volume 42, pages 23–29

Continuous display of plasma potassium during cardiac surgery

H.F. DRAKE, T. TREASURE AND B. SMITH

Summary

We have evaluated the use of an intravascular ion-selective electrode for continuous plasma potassium monitoring in patients during and after cardiac surgery. Potassium estimations from the indwelling electrode provided a rapid and sensitive, but only moderately reliable, indicator of potassium as confirmed by blood samples. Several significant changes in the plasma potassium were found to occur in the period studied and using this continuous monitor, the precise time course of these changes was recorded.

Key words

Equipment; ion-selective electrodes.
Ions; potassium.

During the 1970s continuous monitoring of plasma potassium became possible with the development of ion-selective electrodes based on a valinomycin/potassium tetraphenylborate membrane.[1] The validity of measurements of potassium produced by these electrodes has been shown[2] and both animal and patient studies[3,4] have been carried out using electrodes made by the individual research workers in their laboratories. The theoretical advantages of continuous monitoring are that rapid and transient changes, which may be missed by intermittent samples, are recorded and trends in the potassium level may be clearly seen. As changes in plasma potassium may result in dysrhythmias, including resistant ventricular fibrillation, it might be useful to monitor the potassium continuously when rapid fluxes are occurring.

Using laboratory-made electrodes there are serious problems with adequate quality control and sterilisation, but in spite of the theoretical advantages and the proven feasibility of continuous potassium monitoring, no commercially made biosensors have become widely available. We therefore undertook to evaluate a prototype for such a device, produced at present for trial purposes only, in patients undergoing cardiac surgery.

Materials and methods

The electrode

The potassium-selective electrode used (Simonsen and Weel, Sidcup, Kent) includes features developed over a number of years at St Thomas's Hospital. The electrode is sterilised by

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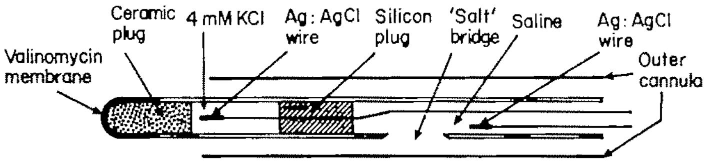

Fig. 1. Components of an ion-selective potassium electrode.

gamma-irradiation and is presented in a pre-sealed pack. Figure 1 shows the principal components of the electrode which is made from 1.0 mm (O.D.) polyvinyl chloride tubing, approximately 20 cm in length, with a valinomycin-based potassium-selective membrane supported by a porous ceramic plug at the distal end. The tubing is filled with an internal reference solution of 4mM KCl and contains two silver/silver chloride electrode wires ('internal' and 'external' reference electrodes). The external reference wire terminates 4 cm from the sensing tip, with a small aperture in the tubing at this point. When placed *in vivo*, the electrode is threaded through an outer cannula containing saline and thus a low-impedance conducting system (or salt bridge) is formed between the sensing and external reference electrodes. The length of the electrode is such that when it is placed correctly, the sensing tip reaches the right atrium. Previous studies in man using similar electrodes for continuous potassium monitoring from the internal jugular vein,[4,5] have used a separate external reference electrode placed outside the body and an internal reference solution of 100mM KCl.

Temperature changes may affect the output from the electrode as the potentials produced by the reference silver/silver chloride electrodes and the potential across the potassium-selective membrane are temperature-dependent. There are two ways of minimising inaccuracies due to temperature fluctuations. Firstly, a balanced design may be used, in which both the sensing and reference electrodes undergo the same temperature fluxes and the potassium activity in the internal reference solution is similar to that in the plasma (i.e. the current design). Alternatively, if the sensing electrode is placed *in vivo* but the external reference electrode is outside the body, a 100mM filling solution is required.

The output from the potassium electrode is passed to a patient-isolated amplifier (Simonsen and Weel) and displayed on a chart recorder (Teckman Electronics).

Patients

Twelve patients undergoing cardiac surgery were included in the study, for which ethical committee approval and signed consent was obtained. Five mitral and four aortic valve replacements and three coronary artery bypass grafting operations were carried out. Six patients were being treated with digoxin and diuretics preoperatively and three had been maintained on cardioselective beta-blocking drugs. Standard anaesthetic and surgical techniques were used. After induction of anaesthesia, a potassium electrode was placed in the right atrium via the right internal jugular vein, through a 14-G cannula (Wallace) using an aseptic technique. The positioning of the electrode was such that a true reading of right atrial potassium, uncontaminated by the coronary sinus effluent, was recorded. A Bentley BOS-10 bubble oxygenator with 1500 ml Hartmann's priming solution was employed. Myocardial protection was induced with 800 ml St. Thomas's Hospital cardioplegia solution containing 20mM KCl; two patients required a further 300 ml and one was given a third dose of 200 ml to maintain cardiac standstill. As this was principally a study to assess the use of this potassium electrode during cardiac surgery, no attempt was made to control rigidly the potassium balance; thus, stored blood was given as required and blood gases and pH were controlled within the limits which are routine for these patients.

The patients were cooled to 27–28°C (nasopharyngeal temperature) at the start of bypass and this temperature was maintained until the cardiac part of the surgery was nearly complete. The design of the potassium electrode was as discussed above such that temperature changes of this magnitude should produce only minimal inaccuracies.

Recordings from the electrodes were made during surgery and for a period postoperatively in the intensive care unit. The electrode response was compared with blood samples taken from an indwelling radial artery cannula at 30-minute intervals during surgery and hourly in the postoperative period; additional samples were taken at the times of peaks or troughs recorded on the continuous trace from the intravascular potassium electrode. The intermittent blood samples were measured immediately on a bench potassium electrode (IL 501, Instrumentation Laboratory, Warrington, Cheshire). This was calibrated before each sample with solutions of known potassium activity and was accurate to within 0.1mM potassium. The arterial site was chosen for sampling both for practical and theoretical reasons. Practically, access to the internal jugular site was unreliable as it was difficult to withdraw blood from the catheter through which the electrode was passed. Also, the removal of blood past the electrode and subsequent flushing with saline solution may cause instability in the continuous recording. Theoretically, it is known both from our own and other studies that the potassium levels may vary between venous sites, in particular, in the proximal inferior vena cava and the right atrium, due to incomplete mixing of streams from different areas of drainage.[6] We therefore considered that the arterial site provided a more constant standard. Ideally, the indwelling electrode would also be placed in an artery, but with the present design of catheter, this might result in unacceptable complications.

Calibration was carried out after removal of the electrode using solutions of known potassium activity and, from the recordings made, the potassium activity in the plasma was calculated by logarithmic conversion.

Results

Practical difficulties with the electrodes included problems with insertion and interference from diathermy during surgery. Three electrodes could not be passed through the cannula in the internal jugular vein and in these patients the study had to be abandoned. The results given thus relate to the remaining nine cases.

Potassium recordings were obtained from the indwelling electrode for 3.5–9.5 (mean 7.1) hours. The comparison of the potassium measured from the blood samples on the bench analyser and the potassium estimation from the indwelling electrode is shown in Fig. 2; the equation for the regression line is $y =$

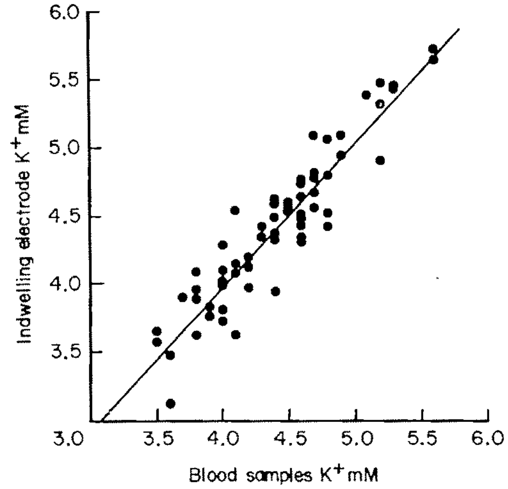

Fig. 2. Regression analysis comparing potassium estimations from intermittent blood samples measured on a bench ion-selective electrode with the potassium recorded from an intravascular electrode. $n = 69$; $r = 0.93$.

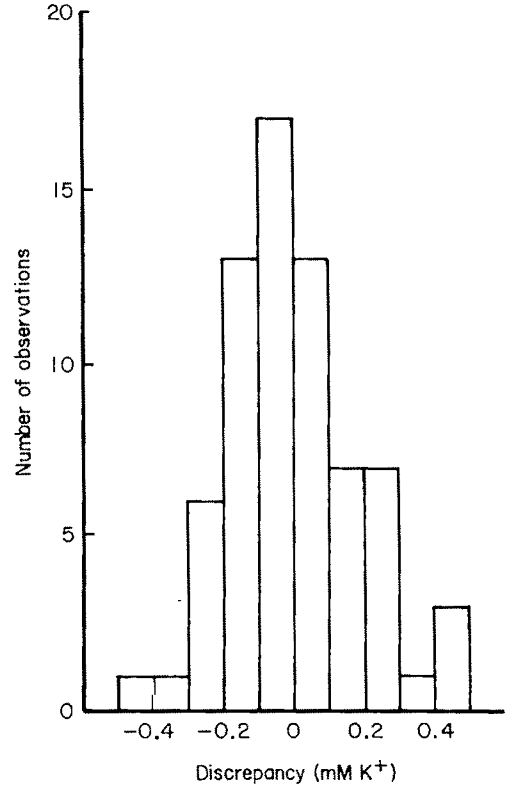

Fig. 3. Histogram to show discrepancies between the potassium estimations from intermittent blood samples and the indwelling electrode.

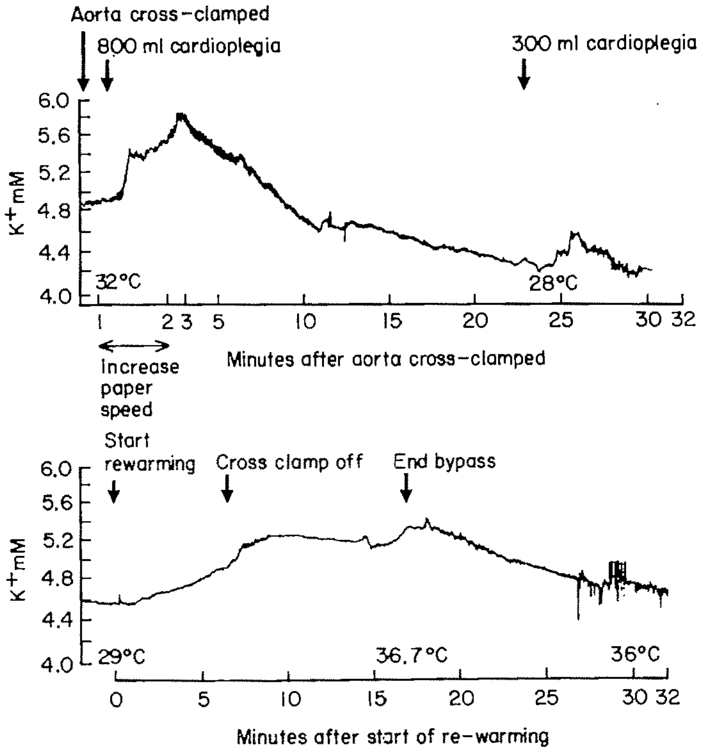

Fig. 4. Examples of recordings of plasma potassium during cardiac surgery. The increase in potassium with the administration of cardioplegia solution and subsequent fall during cardiopulmonary bypass, the rise in potassium during the rewarming phase and abrupt fall at the cessation of bypass are all demonstrated.

$1.05x - 0.21$ and the correlation coefficient (r) was 0.93. The discrepancy frequency histogram (Fig. 3) illustrates that 73% of the results from the electrode fell within 0.2mM of the values of the intermittent samples and the mean (SEM) difference is 0.14 (0.01)mM. On four occasions, the potassium estimation from the indwelling electrode differed from that measured on the bench analyser by more than 0.4mM; the largest discrepancy was 0.49mM.

Examples of the recordings are shown in Fig. 4 and the pooled data for changes in plasma potassium recorded from the indwelling electrode during cardiac surgery are shown in Fig. 5. Control potassium levels postinduction were normal at a mean value of 4.3 (0.12)mM; a transient increase to a peak of 4.8 (0.14)mM occurred as bypass was commenced, followed by a further rise (maximum 5.7 (0.18)mM) when the first dose of cardioplegia solution was given. These values were validated by analysis of withdrawn samples; in particular, the peaks from the continuous tracing after administration of cardioplegia were confirmed by blood samples measured on the bench electrode and thus could not be attributed to artefact as a result of positioning the indwelling electrode at the outflow of the coronary sinus. Initially during bypass, the plasma potassium fell below control, reaching 3.9 (0.15)mM; this was reversed during rewarming to 4.6 (0.17)mM at the end of bypass, but a further fall occurred beginning immediately after restoration of normal circulation; this resulted in a potassium level of 4.2 (0.13)mM at the end of surgery.

For convenience, the grouped potassium estimations taken from the indwelling electrode at stated points during the operative period have been numbered (Fig. 5). Analysis of variance showed significant changes between the potassium levels ($p < 0.01$). All sequential samples (i.e. 1–2, 2–3, 3–4, etc.) showed significant changes ($p < 0.05$) when analysed as a group by a multiple range test, that of Duncan;[7] 2–3, 3–4 and 4–5 were significant at the 1% level. Samples 2 and 3 were significantly different from

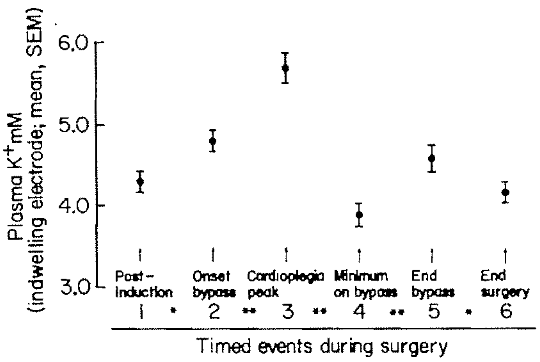

Fig. 5. Mean (SEM) potassium levels (mM) during cardiac surgery measured from an electrode in the right atrium. * and **, significant changes between sequential sampling times at the 5% and 1% levels, respectively (Duncan's multiple range test). Potassium levels at times 2 and 3 were also significantly different from control.

the control values postinduction ($p < 0.05$) with sample 3 also reaching significance at the 1% level. These data are summarised in Fig. 4.

Postoperatively the potassium stabilised, although there was a tendency for a gradual fall and three of the patients were given intravenous supplements to maintain what were considered to be clinically acceptable levels. However, no sequelae which could be directly related to hypokalaemia were seen in the period of study in any of the patients.

Discussion

This study was undertaken to assess a commercially made ion-selective electrode for monitoring changes in plasma potassium during routine surgery. Patients undergoing cardiac surgery were suitable for study because potassium changes are known to occur and there is access to central veins for electrode insertion. The electrodes were relatively simple to use once *in situ*, but a 25% failure rate of correct placement was disappointing. The insertion difficulties seemed to relate principally to the very flexible nature of the electrode; it is possible that this may be improved by the use of stiffer reference wires.

The regression line comparing the potassium estimations from the *in vivo* electrode with those from the intermittent blood samples taken from the arterial circulation had a slope close to unity; this confirms that the catheter electrode response is equivalent to that of potassium electrodes previously described.[2] However, a correlation coefficient of 0.93, which would be excellent for biological data, is not a good test of agreement between two methods of measurement.[8] The discrepancy frequency histogram, which in this instance is almost identical to a histogram of the residuals, relates more readily to familiar clinical measurement.[9] The 73% of results from the indwelling electrode which agreed within 0.2mM with the samples were considered to be within the clinical range of accuracy required. In contrast, the four marked discrepancies of 0.4mM or more would not be acceptable if similar results were seen regularly in the recordings. However, all of these occurred towards the end of the period of monitoring, when the electrodes had been *in situ* for 7–9 hours and with two of these, it was possible to restore the sensitivity of the electrode by flushing the catheter with a bolus of saline. One source of electrode drift is contamination of the membrane by protein deposition and this might be reduced with a slow continuous flush of heparinised saline.

The study confirmed that the electrodes were able to monitor transient changes in the potassium level, for example the short-lived increase which occurs at the onset of bypass and which has been recorded by other workers using a continuous technique.[10] However, this and other transients will almost inevitably be missed by conventional studies which employ intermittent blood sampling. This early change is presumed to be due to mixing with the Hartmann's priming solution in the pump (containing KCl 5mM), but is probably of little clinical significance.

The potassium fluxes which occur during

cardiac surgery have been extensively studied, but there are no published accounts of these changes throughout the operative period measured with an intravascular electrode. Early work[11,12] demonstrated hypokalaemia post-bypass. Increased excretion of potassium in the urine, thought to be due in part to the use of mannitol in the priming solution, and redistribution of potassium into cells, were cited as possible causes. Pre-operative diuretics were also considered as contributing to hypokalaemia, but the evidence for this remains inconclusive. More recent studies have highlighted possible hyperkalaemia occurring at the end of the bypass period, in particular in patients with glucose intolerance, or those on noncardioselective beta-blocking drugs preoperatively.[13,14] Continuous monitoring has been carried out with potassium electrodes in the cardiopulmonary bypass circuit,[10,15] and in the internal jugular vein.[4,5] Using intravascular monitoring, Lim *et al.*[16] have demonstrated a significant increase in potassium during rewarming and have suggested that excessive use of potassium supplements at this time may produce hyperkalaemia.

Our present study group is small, but our results showed several significant changes in the plasma potassium during cardiac surgery. The increase at the onset of bypass has been confirmed and the rise after cardioplegia is to be expected if the coronary sinus return is allowed to drain to the venous reservoir. This was the case in our patients. The decline in potassium on bypass is thought to be related to hypothermia causing movement of potassium into cells[17] and the rise during rewarming is presumed to result from the reversal of this intracellular movement. The magnitude of this increase in our patients was very similar to that reported by Lim *et al.*[16] and it seems important to emphasise that care should be exercised in giving potassium supplements intravenously at this stage in the operation.

It is interesting that when using continuous monitoring, the decrease in potassium which occurs at the end of bypass can be seen to start immediately on cessation of the extracorporeal circulation. However, the cause of this decline in potassium is not clear. It is possible that it is due to improved blood flow to the tissues with the resumption of true physiological perfusion; alternatively, catecholamine levels are known to be elevated after bypass[18] and may contribute to this change.[19]

This preliminary study indicates that the use of a continuous potassium monitor for research purposes during cardiac surgery is of value to characterise more accurately the potassium changes which occur and thus to rationalise the use of intravenous supplements. However, our view remains that the technical difficulties and the limited reliability of the method are such that there is no justification for its use in routine clinical monitoring.

Acknowledgments

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The prevalence of serological markers for hepatitis B virus infection amongst anaesthetists in the Oxford region

M. E. SINCLAIR, M. W. ASHBY AND J. B. KURTZ

Summary

A total of 125 anaesthetists from nine hospitals within the Oxford region were surveyed to study the prevalence of serological markers for hepatitis B virus (HBV) infection. No anaesthetists were positive for Hepatitis B Surface Antigen (HBsAg) and only four (3.2%) were positive for HBsAg antibody (anti-HBsAg). This result is in marked contrast to other studies and suggests that anaesthetists in the United Kingdom do not constitute a high risk population. The reasons for this are discussed.

Key words

Complications; hepatitis B virus infection.

Two recent papers have demonstrated an increased incidence of hepatitis B virus (HBV) infections amongst anaesthetists compared with the general population.[1,2] It is of particular concern that in the USA up to 21% of anaesthetic personnel had antibody to HB surface antigen (anti-HBsAg) and had therefore been infected at some time with HBV and that 2–3% were carriers of HBV at the time of testing. This compares with a cross-sectional prevalence of 18.5% anti-HBsAg positivity in medical staff in the USA.[3] In the UK, the prevalence of anti-HBsAg among hospital staff and the general population has been reported at 7%.[4,5] The recent availability in this country of a safe and effective vaccine against HBV makes the vaccination of high risk groups a feasible proposition.[6,7] This study was initiated to find out if anaesthetists constitute a high risk group.

Subjects and methods

At the time the study was initiated, 181 anaesthetists were based at nine different hospitals within the region. The range of work encompassed by these hospitals includes renal units, a haemophiliac centre and several casualty units, as well as all the major specialties. It was not possible to screen all anaesthetists, because a proportion of those approached declined to participate. The main reason for this was their anxiety that they might have contracted HBV in the past and could therefore be carriers of the virus. Simply excluding these individuals from the study might have resulted in an artificially low figure for the incidence of HBV. To avoid this bias, a single refusal led to the exclusion of the remaining anaesthetists at that hospital. Individuals were also excluded if they had previously

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been vaccinated against HBV, or were repeatedly absent when their hospital was visited. Blood samples were taken from the remaining anaesthetists and examined for HBsAg by direct passive haemagglutination (Hepatest, Wellcome Reagents Ltd) and for anti-HBsAg by passive haemagglutination (Serodia, Fujirebio Inc.). A coded system was used so that neither the investigators nor the laboratory staff would be able to link any result to a particular individual.

Results

A total of 166 anaesthetists were approached to take part in the study; 15 were unobtainable at the time of the study. Four were excluded as they had been vaccinated against HBV and a further 15 declined to take part in the study, thereby excluding two hospitals and 37 personnel. Of the remaining 125 anaesthetists, four (3.2%) were positive for anti-HBsAg. None was positive for HBsAg. The range for years of practice in anaesthesia was 3 months to 40 years, with a mean of 15 years. There was no correlation between years spent in anaesthesia and seropositivity.

Discussion

Our results of a 3.2% prevalence of serological markers for HBV amongst a group of anaesthetists is the lowest yet recorded. The reasons for this low prevalence are not entirely clear, but are probably multifactorial. It is possible that anaesthetists who refused to participate in the study did so because they knew that they were seropositive. This seems unlikely, but even if it were true, our policy of excluding the other anaesthetists at that hospital would negate this bias. Our sample of 125 anaesthetists tested may be small, but represented almost 70% of all the anaesthetists in one of the largest health regions in the UK.

We did not test for Hepatitis B Core antibody (anti-HBc) and the technique of passive haemagglutination for detecting HBsAg and anti-HBsAg is slightly less sensitive than the radioimmunoassay method (RIA) used in the American reports,[1,2] thus slightly reducing our seropositive pick up rate. The same screening technique of passive haemagglutination for detecting both HBsAg and anti-HBsAg has been used routinely at the North London Blood Transfusion Centre and is 6% less sensitive than RIA.[8] Even allowing for this reduction in sensitivity, our finding of a 3.2% incidence of serological markers for HBV is still remarkably low.

The regular testing of staff and patients in renal dialysis units and the isolation of HBsAg-positive individuals has, in the UK, resulted in an overall reduction in the prevalence of hepatitis B virus in patients and the incidence of hepatitis among the staff.[9] However, attempts to reduce the transmission of HBV by identifying carriers through routine review of the notes or patient questioning have been unsuccessful and it is likely that it is the unsuspected, asymptomatic patient that transmits HBV.[1,2]

Hepatitis B virus is not readily transmitted, except from HBV e-antigen (HBeAg)-positive individuals. Asymptomatic HBV carrier mothers (HBsAg-positive) are highly unlikely to transmit HBV to their infants if they are serologically positive for antibody to e-antigen (anti-HBe).[10]

This implies that HBV carriers (HBsAg-positive) with anti-HBe are much less likely to transmit the disease than similar carriers who are HBeAg-positive. In the UK, the prevalence rate of HBsAg carriers in the general population is low and has been estimated as 0.1–0.2%; of these, anti-HBe-positive carriers are about four times more frequent than HBeAg-positive ones.[11]

The annual incidence of acute HBV infection in surgeons in the UK has been estimated at 0.04–0.05%, which is much lower than the reports of past infection in Danish or American surgeons might suggest.[12] However, it is still ten times the reported incidence of acute HBV infection in the general population.[13] Again, surgeons had the highest anti-HBsAg prevalence rate among medical staff in the USA and so it is likely that, in the UK, the prevalence among anaesthetists would also be lower than among UK surgeons.[3] The lowest prevalence of HBV seropositivity in American anaesthetists at one centre was 8.7%, which is still over twice as high as in our study. There are no major differences in anaesthetic technique or practice between the USA and the UK that would account for this. However, in the USA, the overall HBV carrier prevalence is 0.3–0.4%, which is double that found in the UK; thus, the exposure is greater for our American colleagues.

The present study suggests that anaesthetists in the Oxford region are at no greater risk of contracting HBV infection than the general population and as such do not, contrary to previous suggestions, constitute a high risk group. This is possibly due to the effective screening of high risk patients and the low incidence of healthy carriers in the general population.

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A prospective study of 200 consecutive twin deliveries

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Summary

A prospective study of a consecutive series of 200 twin pregnancies has provided some definitive and some tentative conclusions. It has been shown that in any attempt to assess the value of a form of clinical management, cases in which a nuchal cord was present in either twin must be disregarded and that attention must be paid to the techniques of analgesia/anaesthesia provided for delivery. There is a strong suggestion that the provision of an epidural for either Caesarean section or vaginal delivery is markedly beneficial to the second twin. There is also evidence to suggest that when vaginal delivery is conducted under an epidural, the acid-base status of the second twin is very likely to be as good as, or better than, that of its sibling, whereas that is most unlikely to be so among cases delivered without an epidural. Further benefit is conferred upon both twins if the bearing-down reflex is abolished. Attention is drawn to an apparently increasing frequency of abdominal delivery of the second twin following vaginal delivery of the first and to the consequent demand which this must make upon anaesthetic cover.

Key words

Analgesia; obstetric.
Anaesthetic techniques, regional; epidural.

Twin pregnancies have held a fascination down the ages and in more recent times have provided a particular interest for clinicians working in the fields of obstetrics and perinatology. There is a measure of agreement about some of the aspects of twin pregnancy, such as the associated higher incidence of pre-eclampsia, the increased likelihood of premature delivery with, possibly as a result, a higher perinatal mortality rate, but there is as yet no consensus regarding the appropriate lines of management of twin delivery. As is the case with the delivery of a singleton breech, the uncertainty probably derives in part from an understandable reluctance to conduct a prospective randomised trial of several clearly defined protocols. Thus, the clinician's opinion tends to be swayed by personal experience (often of a very limited nature and frequently somewhat biased by recollection of one disaster or one very fortunate outcome) and by the published reports of the experience of others.

It is unfortunate that in most of the accounts of the management and outcome of twin delivery, there is omitted a detailed reference to two of the most important factors which can influence the condition of the immediate neonates, namely, the

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presence or absence of a nuchal cord and the technique of analgesia or anaesthesia provided during delivery. The objective of the present study was in part to include these two factors in an assessment of the factors which can contribute to the condition of newly-born twins.

Methods

Data relating to all twin pregnancies cared for and delivered in the Birmingham Maternity Hospital from the beginning of 1983 to mid-1985 were accumulated. Note was made of the following: maternal pathology during pregnancy and labour; gestational age at the time of delivery, as assessed by the neonatologists (applying the Dubowitz score) or, in the case of abortion, by the pathologist; birth weight of each infant; presentation of each infant at the time of delivery; presence or absence of nuchal cord in each twin; mode of delivery of each twin. With regard to infants delivered by Caesarean section, the technique of anaesthesia was recorded, as was, in seconds, the uterine incision to delivery interval (UD_1 and UD_2 for the first and second twin, respectively); the stated obstetric indications for abdominal delivery were also noted.

With regard to vaginally delivered infants, note was made of the technique of analgesia provided for labour and delivery and of the time elapsing between the attainment of full cervical dilatation and the complete delivery of each infant. With respect to cases in which an epidural had been provided, record was made regarding whether or not the mother had retained the bearing-down reflex. Whenever possible, a double-clamped loop of umbilical cord was secured from each infant, and the acid-base status of blood from the umbilical artery (UA) and umbilical vein (UV) assessed, using a Corning 175 Automatic pH/blood gas system.

The Apgar minus colour scores at one and 5 minutes, $(A-C)_1$ and $(A-C)_5$, were recorded for each infant, as was the time to sustained respiration (TSR) and any resuscitative measures applied. Information regarding the subsequent progress of each live-born infant was obtained from the neonatal records and relevant information was obtained from reports of postmortem examinations of all stillbirths and neonatal deaths.

It should be noted that each mother who was to be delivered by elective Caesarean section was offered the choice of general anaesthesia or epidural analgesia. Those who required delivery by emergency Caesarean section were offered an epidural only if there was sufficient time available for one to be either initiated or extended. An epidural for labour was offered to all mothers for whom it was considered an appropriate choice.

Results

The series consisted of 200 pregnancies. The distribution respecting gestational age at delivery, mode of delivery and technique of analgesia/anaesthesia provided relating to 196 of these is summarised in Table 1. Four cases cannot be easily classified. One was a grand-multipara aged 41 years who went into spontaneous labour at 36 weeks. Twin 1 (2.78 kg) delivered spontaneously by the vertex (Apgar minus colour scores 6/8, TSR 2 minutes); the mother had had no analgesia. Subsequently, the placenta was found to be presenting, with twin 2 presenting as a breech. A single-shot epidural was provided, and breech extraction of twin 2 (2.5 kg) performed (Apgar minus colour scores 1/6, TSR 4 minutes). Tracheal intubation was required. Twin 1 developed pneumonia from which it made a good recovery, while twin 2 made uneventful progress.

In a second mother, both twins died *in utero*, one at 28 weeks, and the other at 31 weeks. The cause was the twin transfusion syndrome. In the third mother both twins aborted at 21 weeks with a diagnosis of Rhesus incompatibility.

The fourth mother developed hyperemesis, loss of weight, hypertension and proteinuria. Intrauterine death of one twin occurred at 28 weeks, while the other twin (1.67 kg) was delivered at 33 weeks. These four cases are not included in further discussion, except with reference to perinatal mortality.

Of the 196 cases referred to in Table 1, 9.0% were delivered by elective and 20.5% by emergency Caesarean section. Among those mothers who laboured, 57.9% delivered vaginally under epidural analgesia, 14.2% delivered vaginally with no epidural, 22.4% were delivered by emergency Caesarean section and 4.4% were delivered of twin 1 vaginally and of twin 2 by Caesarean section.

Vaginal delivery

The distribution of presentation of the infants is detailed in Table 2. The associated maternal

Table 1. Distribution, by gestational age, mode of delivery and type of analgesia/anaesthesia, of the 196 cases of twin pregnancy discussed in the text.

Gestational age at delivery	Vaginal delivery		Elective Caesarean section		Emergency Caesarean section		Vaginal 1/ Caesarean section 2	
	Epidural	No epidural	Epidural	General	Epidural	General	Epidural	No epidural
⩾38	33	8	8	1	6	11	2	2
37	25	4	—	1	1	2	—	—
36	15	1	1	1	2	—	—	—
35	8	4	—	—	1	3	—	1
34	9	2	—	3	2	4	—	—
33	—	1	—	—	—	3	—	—
32	5	1	—	1	—	—	1	1
31	5	1	—	—	—	3	—	—
30	—	—	—	—	—	—	1	—
29	1	1	—	—	—	—	—	—
28	—	1	—	1	—	2	—	—
27	1	—	—	—	—	1	—	—
26	2	—	—	—	—	—	—	—
25	1	—	—	—	—	—	—	—
24	—	1	—	—	—	—	—	—
Totals	105	25	9	8	12	29	4	4

Table 2. Distribution of presentation of infants in the series of 188 cases (eight cases in which twin 1 was delivered vaginally and twin 2 by section have been omitted from this consideration) and in the group of 106 cases in which pregnancy had been maintained for at least 37 weeks.

	Elective Caesarean section		Emergency Caesarean section		Vaginal with epidural		Vaginal without epidural		Totals	
	All	⩾37 weeks	All	⩾37 weeks	All	⩾37 weeks	All	⩾37 weeks	All	⩾37 weeks
vx/vx	4	2	11	6	58	38	15	8	88	54
vx/br	5	2	11	5	32	17	6	2	54	26
br/vx	2	0	7	4	9	4	2	1	20	9
vx/trans	1	0	2	2	0	0	0	0	3	2
br/trans	3	3	2	1	0	0	0	0	5	4
br/br	2	1	3	2	6	4	2	1	13	8
trans/vx	0	0	3	2	0	0	0	0	3	2
trans/br	0	0	1	1	0	0	0	0	1	1
brow/vx	0	0	1	0	0	0	0	0	1	0

vx, vertex; br, breech; trans, transverse.

pathology consisted of: moderate pre-eclampsia (5 cases, at 38, 37, 37, 36 and 32 weeks); gestational hypertension (10 cases, 7 at ⩾ 38 weeks, one each at 37, 35 and 34 weeks); diabetes mellitus (37 weeks); twin transfusion syndrome (37 weeks); rhesus incompatibility (36 weeks); intrauterine growth retardation (36 weeks); placental abruption (3 cases, at 29, 26 and 25 weeks); spontaneous rupture of membranes with premature labour (10 cases, one at 35, two at 32, four at 31, one each at 28, 27 and 25 weeks).

The number of pairs with discordant weights, as defined, is detailed in Table 3. If the definition of discordancy had been reduced to ⩾ 200 g, only three additional pairs of infants would have been categorised as of discordant weight. In four cases, twin 1 was at least 1 kg heavier than twin 2.

Among vertex-presenting infants, the incidence of first twin delivery by forceps or ventouse under epidural block was 48.9%; the comparable figure without epidural was 15.0%. The respective incidences for second twins were 56.9% and 18.9%. All first twins presenting by the breech were delivered as assisted breeches, whether the mother had an epidural or not. With respect to second twins presenting by the breech, 29.0% delivered under epidural were breech extractions, as were 25.0% of those whose mothers did not have an epidural.

The mean interval between full cervical dilata-

Table 3. Analysis of discordant weights. One twin is recorded as being heavier than the other if the difference exceeded 250 g. The numbers in which the difference exceeded 500 g are bracketted.

	1>2	2>1	Equal
Elective Caesarean section	6 (2)	3 (1)	8
Emergency Caesarean section	12 (5)	5 (1)	24
Vaginal delivery	33 (13)	28 (8)	69
Vaginal 1/Caesarean section 2	2 (0)	2 (0)	4

Table 4. Interval from the time of full cervical dilatation to delivery of twin 1 related to (a) the gestational age and (b) the birthweight of the first twin, categorised with respect to whether or not the mother was in receipt of an epidural.

(a)	Epidural			No epidural			
Gestational age (weeks)	*n*	Mean interval (minutes)	Range (minutes)	Gestational age (weeks)	*n*	Mean interval (minutes)	Range (minutes)
⩾38	33	107.6	16–273	⩾38	8	22.5	5–74
37	25	83.0	10–249	<38	17	31.4	5–120
36	15	71.0	8–151				
<36	32	60.4	8–184				

(b)	Epidural			No epidural			
Birthweight (kg)	*n*	Mean interval	Range	Birthweight (kg)	*n*	Mean interval	Range
>3.0	13	76.3	16–209	>3.0	3	9.0	5–17
>2.5–3.0	31	103.5	21–249	>2.5–3.0	8	27.7	6–60
>2.0–2.5	39	84.3	8–273	>2.0–2.5	7	39.2	5–89
>1.5–2.0	15	53.4	10–130	>1.5–2.0	3	13.3	5–22
>1.0–1.5	3	53.0	8–90	>1.0–1.5	4	41.5	9–120
⩽1.0	4	39.2	18–54				

Table 5. Interval between twin deliveries related to (a) the gestational age and (b) the birthweight of the second twin, categorised with respect to whether or not the mother was in receipt of an epidural.

(a)	Epidural			No epidural			
Gestational age (weeks)	*n*	Mean interval (minutes)	Range (minutes)	Gestational age (weeks)	*n*	Mean interval (minutes)	Range (minutes)
⩾38	33	11.3	4–30	⩾38	8	11.6	2–24
37	25	12.2	3–40	<38	17	8.2	3–24
36	15	12.3	3–26				
<36	32	11.4	2–56				

(b)	Epidural			No epidural			
Birthweight (kg)	*n*	Mean interval	Range	Birthweight (kg)	*n*	Mean interval	Range
>3.0	15	13.7	4–33	>3.0	3	11.7	6–22
>2.5–3.0	27	13.0	5–27	>2.5–3.0	8	9.7	[illegible]–24
>2.0–2.5	35	11.6	3–52	>2.0–2.5	4	11.5	3–24
>1.5–2.0	22	11.3	4–56	>1.5–2.0	7	7.7	4–12
⩽1.5	6	7.3	2–13	⩽1.5	3	7.0	6–9

tion and delivery of the first twin under epidural analgesia increased steadily with increasing maturity (Table 4). The relationship between this interval and the birthweight of the first twin was not as clearcut in the epidural series, and in the nonepidural series there was again no apparent correlation (Table 4). With regard to the interval between deliveries, the patterns were different (Table 5). There was little evidence that the mean duration was related to gestational age, but in both the epidural and the nonepidural series there appeared to be a strong tendency for the between-twin interval to be increased as the birthweight of the second twin was increased.

Table 6. Between-twin comparisons of vaginally delivered infants of which neither twin had a nuchal cord. The attributes have been considered as being equivalent if the UA pH difference between the twin values was <0.005, the $P\text{CO}_2$ difference was <1.0 kPa and the base excess (BE) difference was <0.5 mmol/litre.

	Epidural			No epidural		
	1 > 2	2 > 1	=	1 > 2	2 > 1	=
pH	27	19	2	8	1	1
$P\text{CO}_2$	12	8	28	6	—	4
BE	22	18	8	6	3	1

A nuchal cord was present at the delivery of 21.3% of vertex-presenting first twins and 23.5% of vertex-presenting second twins. The respective incidences among infants presenting by the breech were 10.5% and 15.2%. In 4% of cases a nuchal cord was present in both twins.

Comparisons were made of the biochemical status of the pairs of twins, excluding all sets in which at least one twin had a nuchal cord (Table 6). It is striking that whereas in the nonepidural group the acid-base status of twin 2 was infrequently superior to that of twin 1, in the epidural series the acid-base status of twin 2 was almost as frequently superior to that of twin 1 as the converse. As a reflection of this, for example, the mean values of pH in UA blood in the epidural series were 7.300 for twin 1 and 7.296 for twin 2, whereas the respective values in the non-epidural series were 7.306 and 7.227.

There was some evidence that protraction of the interval between twin deliveries was associated with a less favourable outcome for the second twin. In five cases in the epidural series the interval exceeded 15 minutes (20, 22, 30, 33 and 52 minutes). In three of these, the UA base excess of twin 1 was superior to that of its sibling, the values being approximately equivalent in the other two. In both comparable cases in the non-epidural group (the interval in each was 24 minutes), the acid-base status of twin 1 was superior.

Elective Caesarean section

Of the 17 elective Caesarean sections, nine were delivered under an epidural. The prime indications for delivery by elective Caesarean section were moderate pre-eclampsia (6), gestational hypertension (1), malpresentation (7), previous Caesarean section (2) and small pelvis (1).

The distribution of presentation of the infants is detailed in Table 2. The presence or absence of a nuchal cord was recorded too infrequently to permit a realistic review. A pair of twins was designated as being of discordant weights if the difference between their birthweights was at least 250 g. This occurred in 53% of the cases (Table 3).

Eight cases conformed to the definition of 'clinically acceptable ideal', that is, there was no evidence indicative of placental dysfunction, and seven of these received an epidural. Of these latter, all except one set of twins had Apgar minus colour scores of 8/8—one twin 2 scored 7/8. The mean of the UD_1 intervals was 60 seconds (range 20–125 seconds) and of the UD_2 intervals 141 seconds (range 80–190 seconds).

The mean values (and ranges) of the acid-base status of umbilical artery (UA) blood samples for twin 1 were: pH 7.329 (7.303–7.400): $P\text{CO}_2$ 6.69 kPa (5.20–7.37); base excess −3.3 mmol/litre (−0.9– −5.2). The corresponding values for twin 2 were: pH 7.315 (7.263–7.350); $P\text{aCO}_2$ 6.78 kPa (6.20–7.43); and base excess −3.2 mmol/litre (−0.3– −5.9). The individual values did not vary in accordance with the duration of the uterine incision to delivery intervals.

The clinical status and biochemical attributes of each infant were compared with those of its twin, with the exception of one set, in which one twin was an intra-uterine death. The comparisons were drawn on the basis of arbitrary values of distinction (Table 7). The results from this small series suggest that, respecting clinical conditions, under general anaesthesia there is a spread of favoured twin, whereas under epidural the twins are almost invariably of equal status; with regard to biochemical status, twin 1 tends to exhibit less metabolic acidosis than twin 2.

Among the infants delivered under epidural, one twin 1 (36 weeks) was diagnosed as having

Table 7. Comparisons of twin 1 with twin 2 in the series of elective and emergency Caesarean sections conducted under either general anaesthesia or epidural analgesia. $(A-C)_1$ and $(A-C)_5$ denote Apgar minus colour scores at 1 and 5 minutes, respectively. For comparison purposes the attributes have been considered as being equal if: $(A-C)_1$ 8/7 or 6/5/4 or 3–0; $(A-C)_5$ 8 or 7 or <7; time to sustained respiration 0 or $\geqslant 1$; pH difference <0.005; $P\text{CO}_2$ difference <1.0 kPa; base excess difference <0.5 mmol/litre.

	$(A-C)_1$			$(A-C)_5$			Time to sustained respiration			pH			$P\text{CO}_2$			Base excess		
	1>2	2>1	1=2	1>2	2>1	1=2	1>2	2>1	1=2	1>2	2>1	1=2	1>2	2>1	1=2	1>2	2>1	1=2
Elective section																		
General	2	2	3	2	2	3	1	2	4	5	2	0	1	1	5	3	1	3
Epidural	0	0	9	0	0	9	1	0	8	5	1	1	1	0	6	4	2	1
Emergency section																		
General	9	3	15	6	2	19	12	2	13	11	8	1	2	3	15	6	7	7
Epidural	0	0	12	0	0	12	0	0	12	5	5	1	3	0	8	4	0	7

Note that the table excludes reference to three Caesarean sections (one elective, two emergency) conducted under general anaesthesia in which one twin had died before section was undertaken. Also, acid-base data were not available for all cases.

lung fluid and one twin 2 (34 weeks) developed pneumonia, from which it made a good recovery. Among those delivered under general anaesthesia, a pair of twins (34 weeks) with Rhesus disease required exchange transfusions and one twin 1 (28 weeks) suffering from twin transfusion syndrome, required an exchange transfusion. All the live-born infants survived in good health.

Emergency Caesarean section

Of the 41 cases of emergency Caesarean section, 11 were delivered under an epidural. The prime indication for delivery of infants by emergency Caeserean section was malpresentation (14). Other indications were fetal distress (8), pre-eclampsia (5), poor progress in labour (5), cord presentation (4), placental abruption (3), previous Caesarean section (3), Rhesus incompatibility (1) and relative infertility (1); the latter two were destined for elective sections.

The distribution of presentations is detailed in Table 2. The incidence of discordant weights, as defined, was 41.5% (Table 3). By definition, no case in this series satisfied the attributes of the clinically acceptable ideal case. The mean values of the uterine incision to delivery intervals under general anaesthesia were twin 1, 46.7 seconds (range 22–90 seconds), twin 2, 121.2 seconds (40–246 seconds); under epidural the mean values were twin 1, 64.4 seconds (25–105 seconds), twin 2, 124.85 seconds (70–230 seconds).

The between-twin comparisons are detailed in Table 7. Under general anaesthesia the dominant tendency is for the clinical condition of twin 1 to be better than or equal to that of twin 2, whereas under epidural analgesia the clinical status of the twins is always equal. Under general anaesthesia there was no well-defined dominance of biochemical status, whereas under epidural analgesia twin 1 exhibited a metabolic acidosis which was either less pronounced than, or approximately equal to, that of twin 2. The distinction was not related to the time elapsing between the two deliveries. Under general anaesthesia, the mean time was 92 seconds (range 60–186 seconds) when twin 1 was biochemically superior to twin 2, and 77 seconds when twin 2 was biochemically superior to twin 1. The corresponding figures for the epidural mothers were 59 seconds (range 18–110 seconds) and 55 seconds (range 15–102 seconds), respectively. Details of the progress of the neonates are presented in a subsequent section.

Vaginal delivery of twin 1, abdominal delivery of twin 2

There were eight cases in this group. None had discordant weights as defined. The indications for Caesarean section were: transverse lie, cervix retracted to 4 cm dilatation (38 weeks, 2.63 kg, no epidural, interval between twin deliveries 8 minutes); breech with prolapsed cord (38 weeks, 3.16 kg, epidural, interval 21 minutes); brow presentation and 'maternal irritability' (40 weeks, 2.18 kg, no epidural, interval 39 minutes); cord prolapse and compound presentation (32 weeks, 1.72 kg, epidural, interval 18 minutes); transverse lie (35 weeks, 2.30 kg, no epidural, interval 81 minutes); failed breech extraction, fetal distress (32 weeks, 1.60 kg, epidural, interval 19 minutes); prolapsed arm, shoulder impaction (30 weeks, 1.60 kg, epidural, interval 19 minutes); footling breech diagnosed, attempted version under general anaesthesia, hand presentation found at Caesarean section (39 weeks, 2.94 kg, epidural, interval 51 minutes).

The second twins of the first, third, fourth, sixth and eighth mothers were in good condition at birth and remained well. The second twin of the second mother was mildly depressed with Apgar minus colour scores of 4/8, but progressed well. The second twin of the fifth mother had Apgar minus colour scores of 3/4 and a TSR of 11 minutes. It developed the respiratory distress syndrome but made a good recovery. The remaining infant scored 2/6 with a TSR of 7 minutes and was extensively bruised, but made a good recovery.

The clinically acceptable ideal case

For the purposes of the present discussion the attributes of cases in this category are no evidence suggestive of placental dysfunction, no nuchal cord and a gestational age of at least 38 completed weeks. Understandably, the number conforming to this definition which emerges from a series even as large as 200 deliveries is quite small, but it can only be from an analysis of such highly selected cases that reliable information about certain important aspects of clinical care can be derived.

The distributions of Apgar minus colour scores and of times to sustained respiration are detailed in Table 8. The paucity of data precludes firm conclusions, but it can certainly be seen that infants presenting by the breech—whether first or second twin—tend to be in poorer clinical condition at delivery than do those presenting by the vertex, irrespective of whether or not an epidural had been in progress. There is a slight suggestion that vertex-presenting twins tend to be in a better clinical condition when the mother is in receipt of an epidural than otherwise. With respect to mothers in the epidural series, whether or not the bearing-down reflex had been abolished appeared not to influence the pattern depicted in Table 8.

A sample of UA blood was obtained from only a selection of these cases, due to either technical or administrative difficulties. Analysis of the results is presented in Table 9. Clearly, the paucity of data precludes any attempt at statistical analysis. However, some tendencies can be tentatively distinguished. When an

Table 8. Distribution of Apgar minus colour scores at 1 and 5 minutes and of time to sustained respiration among CAI infants as defined, related to mode of delivery and the provision of an epidural for labour. There were no breech-presenting first twins in the nonepidural group.

	$(A-C)_1$			$(A-C)_5$			Time to sustained respiration	
	8/7	6/5/4	3–0	8	7	<7	0	≥1
Twin 1 vertex								
Epidural	21	0	0	21	0	0	21	0
No epidural	7	0	0	7	0	0	6	1
Twin 2 vertex								
Epidural	9	1	0	10	0	0	9	1
No epidural	2	1	0	3	0	0	1	2
Twin 1 breech								
Epidural	1	4	0	5	0	0	0	5
Twin 2 breech								
Epidural	2	5	0	6	1	0	3	4
No epidural	1	0	1	2	0	0	1	1

Table 9. Mean values of UA pH, $P\text{CO}_2$ (kPa) and base excess (mmol/litre) in the series of CAI cases, categorised with respect to mode of presentation, whether or not the mother was in receipt of an epidural and, if she was, whether or not she had retained the bearing-down reflex (b/d).

	n	pH	$P\text{CO}_2$ (kPa)	Base excess (mmol/litre)
Twin 1 vertex				
Epidural no b/d	14	7.290	6.49	−6.01
Epidural + b/d	2	7.308	5.90	−6.60
No epidural	5	7.312	5.84	−6.28
Twin 2 vertex				
Epidural no b/d	4	7.305	6.37	−5.65
Epidural + b/d	2	7.237	7.14	−7.50
No epidural	3	7.146	8.11	−10.33
Twin 1 breech				
Epidural no b/d	2	7.325	6.07	−5.40
Twin 2 breech				
Epidural no b/d	4	7.312	6.13	−5.50
Epidural + b/d	1	7.260	7.10	−6.00

Table 10. Distribution of mean values as in Table 9, but including all cases of gestational age at least 28 completed weeks and otherwise comforming to the definition of clinically acceptable ideal cases.

	n	pH	$P\text{CO}_2$ (kPa)	Base excess (mmol/litre)
Twin 1 vertex				
Epidural no b/d	37	7.289	6.42	−5.51
Epidural + b/d	6	7.278	6.04	−7.70
No epidural	8	7.307	5.76	−6.51
Twin 2 vertex				
Epidural no b/d	21	7.285	6.24	−5.41
Epidural + b/d	5	7.288	6.38	−6.70
No epidural	8	7.211	7.10	−9.06
Twin 1 breech				
Epidural no b/d	6	7.271	6.74	−6.33
Epidural + b/d	1	7.260	6.60	−7.00
Twin 2 breech				
Epidural no b/d	14	7.274	6.59	−6.44
Epidural + b/d	3	7.282	6.82	−5.50
No epidural	3	7.239	7.10	−7.30

epidural is in progress, retention of the bearing-down reflex is associated with a greater degree of metabolic acidosis. The second twin tends to be considerably more acidotic than twin 1 if the mother has not received an epidural; this does not pertain among cases in which an epidural was provided. The latter observation applies irrespective of the presentation of each infant.

If a similar analysis is made of a series in which the definition of the clinically acceptable ideal case is extended to include all cases of gestational age at least 28 weeks, the pattern is strengthened (Table 10) and statistical evaluation becomes feasible. A comparison of the mean values relating to vertex-presenting twins delivered under epidural (with or without retention of the bearing-down reflex) with those for twins delivered of mothers who did not receive an epidural reveals the following: twin 1 with epidural versus twin 1 without epidural, no significant differences; twin 2 with epidural versus twin 2 without epidural, pH $p<0.01$, $P\text{CO}_2$ not significantly different, base excess $p<0.001$; twin 1 with epidural versus twin 2 with epidural, no significant differences; twin 1 without epidural versus twin 2 without epidural, pH $p<0.01$, $P\text{CO}_2$ $p<0.01$, base excess not significant. There were no significant differences between twins 1 and 2 delivered under an epidural, whether either or both were presenting as a breech.

The importance attached to the necessity of identifying the presence or absence of a nuchal cord when conducting investigations of this order can be demonstrated by comparing the acid-base values relating to the vertex-presenting infants delivered under epidural referred to in Table 10 with those from a group of infants similar in all respects except that they were delivered with a nuchal cord. Among first twins (13 infants), the mean values of pH and base excess among infants with a nuchal cord (7.246 and −7.92 respectively) differed significantly from those without a nuchal cord ($p<0.05$ in each instance). Among second twins (12 infants), the mean values of all three biochemical variables were statistically significantly different: pH 7.214 ($p<0.01$), $P\text{CO}_2$ 7.54 kPa ($p<0.01$) and base excess −8.01 mmol/litre ($p<0.05$). A similar distinction could be identified with respect to the clinical condition of these infants.

Neonatal progress

All the live-born infants delivered by emergency Caesarean section survived, except for a second twin (35 weeks) delivered under general anaesthesia, who succumbed to necrotising enterocolitis. Three were temporarily embarrassed by lung fluid: twin 2, 38/52, twin 1, 34/52, and twin 1, 31/52, each delivered under general anaesthesia. Twelve infants developed the idiopathic respiratory distress syndrome: delivered under epidural: twin 1, 38 weeks (also diagnosed as evidencing intra-uterine growth retardation), twin 1, 34 weeks and twin 2, 34 weeks; delivered under general anaesthesia: twin 1, 31 weeks, three second twins 31 weeks, twin 1, 28 weeks, two second twins at 28 weeks, twin 1, 27 weeks and twin 2, 28 weeks.

A second twin of 35 weeks delivered under general anaesthesia developed a coagulopathy and renal failure, and recovered. In addition, a persistent ductus arteriosus was diagnosed in two infants, coarctation of the aorta in one and Down's syndrome in another. Several of the more immature infants exhibited recurrent apnoeic spells. All vaginally delivered infants of gestational age at least 38 weeks at delivery survived without mishap.

Of the 10 infants delivered at 27 weeks gestation or less, only two survived: a first twin (27 weeks, epidural) who recovered from an episode of necrotising entercolitis and a first twin (25 weeks, epidural) who survived the respiratory distress syndrome and some of its attendant ills (intraventricular haemorrhage, convulsions and a patent ductus arteriosus). The death of two of the infants was related to gross immaturity (24 weeks, no epidural) and the remaining deaths were associated predominantly with respiratory distress syndrome. All of these latter six infants were delivered under an epidural: two first twins at 26 weeks, two second twins at 26 weeks, one second twin at 27 weeks and another second twin at 25 weeks.

Twelve of the 118 infants of gestational age 28–36 weeks developed respiratory distress syndrome. There is a suggestion that the complication tends to be higher among second twins than among their siblings, the incidences being: twin 1, epidural 7.9%, no epidural 9.1%; twin 2, epidural 13.2%, no epidural 27.3%.

Discussion

Despite the fact that this is a detailed analysis of quite a large series of twin deliveries, it has afforded very few firm conclusions, attesting to the difficulties which face clinicians who are required to make decisions relating to the care of patients in this category. It is only when the results of similar analyses of even larger series become available that soundly based guidelines will begin to replace intuition and the prejudice of occasional experience. However, even from the present study, some tentative conclusions, which either confirm or question current opinion, can be drawn.

In 19.6% of cases, both twins were delivered by Caesarean section. This compares with an incidence of 15.1% Caesarean sections among singleton pregnancies in this hospital during the same period. Of greater interest, however, was the observation that in 4.4% of cases the second twin was delivered by section following vaginal delivery of the first twin (in 3.3% of cases in which an epidural was provided and in 6.8% of those in which it was not). The present writer must confess that he found this to be both novel and somewhat alarming. However, recently published reports suggest that it is no longer such an unusual practice as it was in the past. Although Leysen *et al.*,[1] in the course of a case report, declared that they had found reference to only 36 cases in the recent literature, Rayburn *et al.*,[2] who reported an incidence of 9%, suggest that if the delay between twin deliveries is greater than 15 minutes, approximately 18% of the second twins will be delivered by section. Eskes *et al.*[3] reported that of their series of 54 cases in which twin 1 delivered vaginally, the second twin was delivered by section in two (3.7%) and in a preliminary report of a Scottish twin study[4] it is noted that 19 second twins were delivered by section following the vaginal delivery of the first twin, an incidence of 3.9%.

Evrard and Gold[5] presented data referable to four such cases in their consecutive series of twin pregnancy, representing 2.3% of the cases in which the first twin was delivered vaginally. They also made reference to five other cases of which they had knowledge and noted that there were two perinatal deaths in this group of nine pregnancies. Chervenak *et al.*[6] reported that in a series of 135 labouring women under their care, with the first infant presenting and being delivered by the vertex and the second either as a breech or transverse, five (3.7%) of the latter were delivered by section. It would be interesting to know if this is a recent phenomenon and, if so, what has prompted its emergence. It is arousing concern in several quarters.[7] Certainly, the prospect of such an event adds further substance to the insistence that a well-prepared anaesthetist must be in attendance at every twin delivery. It might even soon become advisable that all intended vaginal deliveries of multiple pregnancy be conducted in the operating theatre.

The relatively high incidence of forceps delivery, of first and second twins, associated with an epidural is unremarkable in the light of current obstetric practice (which is not necessarily to be condoned). It is, however, pleasing to note that in contrast to experience previously reported from this hospital,[8] the incidence of breech extraction is not closely related to the provision of an epidural.

The period between the estimated time of achievement of full cervical dilatation and vaginal delivery of twin 1 was much longer in the epidural series than in the remainder. Interestingly enough, as detailed above, in the epidural group there was a direct relationship between the length of this interval and gestational age (a relationship which was not reflective of a varied incidence of instrumental deliveries), but a similar distinction was not to be seen in the nonepidural group, and the relationship between this interval and the birthweight of twin 1 was not as distinct.

The mean interval between twin deliveries was closely similar in the epidural and nonepidural groups, although the range in the former was considerably greater. The mean duration of this interval was not apparently related to gestational age, but did appear to be increased in accord with an increase in the weight of twin 2.

It is of considerable interest that the biochemical status at delivery of twin 2 was almost as frequently equivalent or superior to that of twin 1, as was twin 1 superior to twin 2, when the delivery was conducted under epidural block. The number of cases in the nonepidural series is small, but the data available support the accepted contention[3] that when an effective epidural is not in progress the condition of twin 2 tends to be less favourable than that of its sibling. On the other hand, Young *et al.*[9] observed that when the interval between twin deliveries was 5 minutes or more, the clinical and biochemical superiority of twin 1 over twin 2 became well established and in the discussion which followed their presentation, they claimed that this was to be seen in cases in which an epidural was in progress. One wonders whether the mothers involved were exposed to aortocaval compression during the period of delivery.

Examination of data from the only cases which can realistically provide the facility of comparisons in the field (the clinically acceptable ideal cases) reveals that irrespective of whether the infants were delivered by the vertex or by the breech, second twins delivered under an epidural are in significantly better condition than those delivered of mothers without an epidural. Although the first twins in the epidural series tended to be in better biochemical condition than those in the nonepidural series, the difference was not statistically significant. An additional observation is worthy of note. The condition of infants delivered under an epidural and whose mothers did not retain the bearing-down reflex was superior to that of infants whose mother did retain the reflex. In this context observations about the data included in Tables 9 and 10 are possibly advisable. Although the mean pH value derived from samples of UA blood in the nonepidural series is higher than those from each epidural group, that undoubtedly reflects the maternal hypocarbia induced by hyperventilation. The relatively greater metabolic acidosis in the 'bearing-down' group compared with that in the series of cases in which the bearing-down reflex was obtunded is most likely reflective of an infusion acidosis consequent upon maternal exertion.[10]

Thus, although the provision of an epidural is associated with a longer interval between full cervical dilatation and delivery, and with a higher incidence of operative deliveries, the condition of both twins is superior to that of twins delivered of mothers who had not been in receipt of an epidural. They are, however, undoubtedly more metabolically acidotic than those in a similar category delivered by elective Caesarean section.

The subsequent postnatal course of infants in this small series of vaginally delivered infants appeared to be uninfluenced by whether or not the mother had received an epidural for labour and delivery.

Finally, attention must be firmly drawn to the potential influence of a nuchal cord upon the condition of the infant. No realistic comparative assessment of the influence of different techniques of management of the delivery of twins can be made without excluding from consideration cases in which this randomly occurring complication was present.

Conclusions

Several major lessons can be drawn from this study. Even a 3-year study conducted in a hospital caring for over 5000 deliveries annually does not afford sufficient information to provide for many firm conclusions regarding the management of twin deliveries. To pursue such a study for much longer would, however, invoke the potential distortion of changing clinical policies. On the other hand, a multicentred survey invites the possibly even greater distortion of slight but significant differences which escape detection. The provision of an epidural for a labouring woman with a twin pregnancy is to the advantage of the infants. This advantage is increased if the maternal bearing-down reflex is obtunded. In the absence of fetal distress, there should be no impetus to attempting to foreshorten the interval between twin deliveries when these are conducted under an epidural and the mother is guarded against aortocaval compression. Infants with the randomly occurring complication of a nuchal cord must be excluded from any scientific analysis of the outcome of twin pregnancies. The sequence of abdominal delivery of twin 2 subsequent to vaginal delivery of twin 1 is not a rarity and arrangements should be made to ensure that it is expeditiously managed should the situation arise.

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Anaesthesia, 1987, Volume 42, pages 44–48

CASE REPORT

Severe acute anaemia in a Jehovah's Witness

Survival without blood transfusion

P. J. HOWELL AND P. A. BAMBER

Summary

A case is described in which a Jehovah's Witness underwent emergency surgery following which her haemoglobin fell to 1.8 g/dlitre. She was successfully treated in an intensive care unit with intermittent positive pressure ventilation of the lungs, high inspired oxygen concentrations and transfusions of large volumes of gelatin solution.

Key words

Blood; loss, gelatin replacement.
Jehovah's Witness.

The moral dilemmas which arise out of the treatment of Jehovah's Witnesses who refuse blood and blood products have been discussed by many authors.[1-4] The clinical management of such patients for elective cardiothoracic[5,6] and major surgery[7-9] has been described, but there is little information about the management of acute blood loss presenting as an emergency.[10-12]

The following describes the postoperative management of an acutely anaemic Jehovah's Witness who required emergency surgery. Blood and blood products would normally have been considered life-saving, but were refused. Despite this, she was successfully managed in an intensive care unit with intermittent positive pressure ventilation and volume replacement.

Case history

A 41-year-old Jehovah's Witness in her sixth pregnancy presented at 32 weeks with obstructive jaundice. Ultrasound showed dilated intrahepatic and common bile ducts. Labour was induced at 35 weeks and she was delivered of a normal, healthy baby girl. Eleven days later, an endoscopic retrograde cholangiopancreatogram demonstrated a stone in the biliary tree and endoscopic sphincterotomy was performed. Unfortunately, bleeding occurred from the sphincterotomy site, which necessitated an emergency laparotomy. Pre-operatively, the haemoglobin was decreased to 9.8 g/dlitre; the prothrombin time was normal. The arterial blood pressure was 80/65 mmHg and the pulse rate was 100 beats/minute. She was conscious but drowsy, after diazepam emulsion 20 mg given for the endoscopy.

After pre-oxygenation, anaesthesia was induced with fentanyl 50 μg and etomidate 4 mg. Cricoid pressure was employed and suxamethonium 100 mg used to facilitate rapid tracheal intubation. Anaesthesia was maintained

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with 40–50% nitrous oxide in oxygen, fentanyl and atracurium. Blood pressure and pulse rate remained satisfactory at 100–110 mmHg systolic and 80–1000 beats/minutes, respectively. Electrocardiographic changes consistent with myocardial ischaemia developed towards the end of the procedure. During the operation she received a total of 2500 ml of gelatin solution (Haemaccel) and 250 ml of 20% mannitol. The total fluid loss was 2050 ml, a mixture of blood, gelatin solution and bile.

Cholecystectomy was performed, one stone was removed from the common bile duct and a large retroduodenal haematoma was evacuated. At the end of the procedure the patient was transferred, anaesthetised, intubated and ventilated, to the intensive care unit. The pulse rate was 110 beats/minute in sinus rhythm, blood pressure 100/60 mmHg and central venous pressure + 3 cmH_2O. The haemoglobin was 3.1 g/dlitre and her clotting screen was within expected limits for the degree of haemodilution.

Discussion with both the patient and her husband before the operation had made it clear that they did not wish either blood or blood products to be administered under any circumstances and a disclaimer was signed to this effect. Despite the patient's critical postoperative state, her husband could not be persuaded to change his views, which were respected.

Discussion with the local Blood Transfusion Service confirmed that there were no synthetic oxygen-carrying substances available in this country. It was therefore decided to continue intermittent positive pressure ventilation to maintain optimal oxygenation and to reduce the risk of pulmonary oedema. Gelatin solution was used to maintain her circulating volume. She was given vitamin K, cimetidine and cefuroxime, with midazolam and fentanyl for sedation and analgesia. Two hours later, although still in sinus rhythm, her blood pressure became unrecordable but was restored after a rapid infusion of gelatin solution.

Postoperatively, in the first 13 hours she received a total of 4750 ml of intravenous fluid, of which 4150 ml was gelatin solution and the balance crystalloid. Over the next 24 hours she received 5565 ml (4950 ml gelatin solution) but she was unable to maintain her blood pressure and therefore on the third day a dopamine infusion was started and increased to a rate of 9 μg/kg/minute to achieve a systolic blood pressure of 100 mmHg. On the fifth day she received 3787 ml (2380 ml gelatin solution), but her blood pressure was still only 70–80 mmHg systolic and her central venous pressure + 10 cmH_2O. She had passed 3330 ml of urine in the preceding 24 hours. It was considered that to increase her fluid input in an attempt to raise her central venous pressure and blood pressure, but without using plasma protein, would put her at risk of developing pulmonary oedema (serum albumin at this time was 10 g/dlitre). An infusion of amino acid and glucose solution (Vamin/Glucose) was started to provide a substrate for the synthesis of albumin. She developed oedema of her hands and forearms and so was started on regular diuretic therapy. The chest X ray remained free from signs of pulmonary oedema.

The dopamine requirement over the next several days increased, so that by the twelfth day it had reached 15 μg/kg/minute. Dobutamine was therefore introduced and the dopamine decreased to 5 μg/kg/minute. The blood pressure gradually increased in the succeeding 24 hours and permitted a slow withdrawal of dobutamine.

After admission to the intensive care unit, her haemoglobin fell to a minimum of 1.8 g/dlitre on the fifth day, and thereafter began to increase steadily (Fig. 1). Other haematological values at that time were haematocrit 0.05, mean corpuscular volume 96 fl and mean corpuscular haemoglobin concentration 34 g/dlitre. The mean corpuscular haemoglobin continued to rise, reaching a maximum of 115 fl on the eleventh day. The mean corpuscular haemoglobin concentration reached a maximum of 39 g/dlitre on the thirteenth day. She was treated with parenteral iron sorbitol and folic acid. Minisamples of blood, 2 ml for urea, electrolytes and liver function tests and 0.5 ml for full blood count, were used throughout to minimise iatrogenic blood loss. Although further haematological investigation would have been of interest, we could not justify the withdrawal of additional samples in a patient with such severe anaemia.

Ventilation of the lungs was maintained with 60% inspired oxygen, and reduced to 50% on day 10. Blood gas analyses were satisfactory throughout; arterial oxygen tension ($Pa{O_2}$) 26–36 kPa, arterial carbon dioxide tension ($Pa{CO_2}$) 3–5 kPa, with a calculated oxygen content of 4–5 ml/100 ml, which gradually increased as her haemoglobin improved. She developed a chest

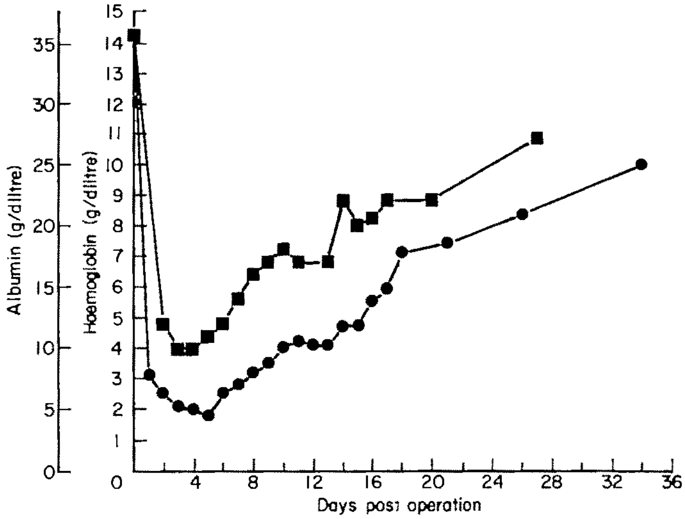

Fig. 1. Serum haemoglobin (●) and albumin (■) concentrations after laparotomy.

infection on the tenth day which was successfully treated with gentamicin. The tracheal tube was removed on the sixteenth day, after a short period of weaning with continuous positive airway pressure.

She developed severe hypernatraemia with a serum sodium concentration of 168 mmol/litre on the sixth day. The sodium content of the gelatin solution is 145 mmol/litre and so the infusion was stopped. Within a few hours her blood pressure had decreased to 70 mmHg systolic and so the infusion was restarted. Within 24 hours the serum sodium started to decrease so that by the eighth day it had reached 156 mmol/litre and by the twelfth day it was 145 mmol/litre. Nasogastric feeding was commenced on the fifth day.

She had periods of pyrexia, up to 39°C and persistent, moderate abdominal pain. On the eighteenth day a sudden increase in the white cell count to 36800/cu mm and the loss of the T-tube drain within the abdomen prompted further surgical intervention. At laparotomy, the drain was recovered and a large collection of bile was drained from the right upper quadrant of the abdominal cavity, from which coliform bacteria were grown.

She returned to the general ward after the operation. The haemoglobin value was 7.1 g/dlitre and albumin 22 g/dlitre. Thirty-four days after the initial surgery, she returned home with a haemoglobin value of 7.7 g/dlitre. Her only permanent deficit was a right vocal cord palsy.

Discussion

Elective surgery without the use of blood transfusion has been performed in orthopaedics,[7,8] gynaecology[9] and cardiothoracic surgery.[5,6] The methods of management include extreme haemodilution,[13] autotransfusion,[1] intra-operative re-infusion[14,15] and controlled hypotension.[7] In the emergency situation, these techniques may be inappropriate because of lack of time to assemble the equipment, as was the case with our patient.

The management of a Jehovah's Witness presents a challenge to the physician who would normally use blood and blood products in the treatment of severe anaemia and hypovolaemia. However, although blood transfusion is refused by most Witnesses, there are a few whose resolve may weaken when faced with the prospect of their impending death[12] and, although undue pressure should not be applied, it is always worth asking the patient or the next of kin to reconsider their decision.

Unfortunately, plasma and albumin solutions are also unacceptable to the strict believer since they have been derived from blood and are similarly forbidden. The choice, therefore, lies between infusions of artificial plasma expanders and crystalloid solutions. In our patient, in the presence of severe hypoproteinaemia, with the attendant risk of pulmonary oedema, the artificial plasma expanders with their greater oncotic pressure were felt to be the fluids of choice.

In severe acute anaemia, oxygen carriage to the tissues is greatly reduced and may result in loss of consciousness or cerebral confusion, oliguria and myocardial ischaemia. The compensatory hyperventilation will itself increase oxygen requirements as will uncontrolled postoperative pain. Maximum oxygen availability must be maintained. Harris *et al.*[10] administered 60% oxygen to their spontaneously breathing patient by facemask. Unfortunately, that patient succumbed. We also used an inspired oxygen concentration of 60%, but in addition our patient's lungs were mechanically ventilated, which ensured maximal oxygenation and also perhaps had other advantages (below). Nearman and Eckhauser[11] ventilated their patient with 100% oxygen, although it is not stated for how long they maintained such a high inspired oxygen concentration.

Oxygen transport to the tissues could be increased by the use of an hyperbaric chamber, but the period of exposure to very high oxygen partial pressures is limited because of toxicity problems and this form of therapy is better suited to the case where further treatment (such as blood transfusion) is envisaged in the near future. By contrast, our patient's problem of severe anaemia was likely to continue for several weeks. The use of a solution of artificial oxygen-carrying molecules (Fluosol DA) was considered, but these substances were not available in the United Kingdom at that time. Blood pressure and cardiac output were maintained in our patient by potent inotropes to ensure adequate tissue perfusion and optimal oxygen delivery under the circumstances.

Nearman and Eckhauser[11] reduced the oxygen consumption of their patient by surface cooling, muscle paralysis, controlled ventilation and a barbiturate infusion. We employed only nonbarbiturate sedation and ventilation. We did not insert a pulmonary artery catheter for risk of haemorrhage and so oxygen consumption could not be measured. Tissue oxygenation was monitored by observation of organ function; urine output, haemoglobin and albumin synthesis, and myocardial performance (assessed by measurement of blood pressure and central venous pressure) and peripheral perfusion were also monitored.

In anticipation of a high rate of haemoglobin synthesis, haematinic agents were given regularly. The average increase in haemoglobin concentration was approximately 2 g/week. This was more than had been expected and may have reflected the normal postpartum haemoconcentration.[15]

Our patient did not develop severe pulmonary oedema, although this was a possible consequence of hypoproteinaemia from severe anaemia. It is likely that positive pressure ventilation was a factor in preventing this. Positive end expiratory pressure was not indicated and it could have worsened her hypotension. She was, however, weaned from the ventilator with a short period of continuous positive airways pressure, to reduce the risk of pulmonary oedema.

The clotting screen reflected serious haemodilution but, surprisingly, there were no problems with bleeding. This may have resulted from the normal persistence of the hypercoagulability of pregnancy into the postpartum period. It has been shown[16] that women can withstand haemorrhage better in the early postpartum period and this may also have contributed to our patient's survival.

In one study,[17] infection was found to be a major complication of severe anaemia. Our patient suffered a chest infection but this is a common complication of prolonged ventilation. In our search of the English literature, we have found no other case of a patient with such a low haemoglobin who has survived without transfusion of blood or artificial oxygen-carrying substances. We believe that she owed her survival to simple intensive care management.

The comparatively new techniqe of endoscopic sphincterotomy has a much lower morbidity and mortality than conventional surgery. However, the major risk is of haemorrhage and a recent review[18] has shown that up to 5% of patients may bleed; of these, one third will require surgery and of these half will die. It is unfortunate that our patient, with her particular religious beliefs, should have fallen into this 5% of patients who bleed.

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CASE REPORT

The neuroleptic malignant syndrome

Case report with a review of the literature

P. ALLSOP AND A. J. TWIGLEY

Summary

Following the administration of fluphenthixol (a depot phenothiazine) for a psychotic illness, a 44-year-old woman developed weakness, rhabdomyolysis and renal failure, together with hyperthermia (42°C) and signs of both autonomic and central nervous system dysfunction. She died following massive intestinal haemorrhage, intra-abdominal sepsis and probable disseminated intravascular coagulation. A diagnosis of neuroleptic malignant syndrome had been made, but treatment with dantrolene sodium was probably instituted too late to prevent the progress of the complications she had developed. This syndrome, which follows the use of phenothiazines or butyrophenones, is rare, potentially fatal and probably underdiagnosed. It has been likened to malignant hyperthermia, but a review of the literature points to many differences. Both dantrolene sodium and dopaminergic drugs (bromocriptine, amantidine and L-dopa) have been shown to be efficacious and their continued use, despite a failure in this case, is advocated until more is known about this syndrome.

Key words

Ataractics, phenothiazines; fluphenthixol.
Complications; hyperthermia.

In 1968 Delay and Denker[1] coined the term neuroleptic malignant syndrome to describe a drug fever associated with neurological and autonomic dysfunction related to phenothiazine treatment. It is a potentially lethal condition and although rare, may not be as uncommon as the paucity of reports suggests. The condition is of interest to anaesthetists, not only because some cases require intensive care, but also due to some similarities with malignant hyperthermia. The following case report illustrates some of the features and potential complications of this condition.

Case history

A 44-year-old female was admitted to another hospital with a 4-day history of generalised weakness, malaise, muscular aches, fever, shivering and a productive cough. A similar episode had occurred in 1982, associated with a positive venogram suggesting a deep vein thrombosis in the leg, for which she was treated with anticoagulants and antibiotics. She had also been diagnosed as having monosymptomatic psychosis and was being treated with a phenothiazine, fluphenthixol (Depixol); her last dose had been 4 weeks prior to the present admission.

On examination she was apyrexial, alert and orientated. Her arterial blood pressure was unrecordable, pulse regular at 120 beats/minute and there was marked vasoconstriction. She fainted when she sat up, but further examination was normal except for a few left basal crepi-

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tations. Initial investigations revealed a leucocytosis (14 000 cells/cu mm) but the haemoglobin, platelets, urea and electrolytes and blood gases were normal. Her urine was strongly positive for blood and protein (she was not menstruating), but microscopy showed only a few white cells. The ECG and chest X ray were also normal.

A provisional diagnosis of septic shock was made and after the insertion of a pulmonary artery catheter, treatment commenced with intravenous colloids and crystalloids. This raised the central venous pressure from -2 to $+8$ cm H_2O, but the hypotension and tachycardia remained, which suggested a primary myocardial aetiology even though serial ECGs remained unchanged. There was some response to inotropic (dobutamine) and vasodilator (sodium nitroprusside) treatment, but she suffered a respiratory arrest and required tracheal intubation and intermittent positive pressure ventilation (IPPV).

She remained hypotensive, with a systolic arterial pressure of 60–70 mmHg, central venous pressure of $+5$ to $+8$ cm H_2O and a pulmonary capillary wedge pressure of 12–15 mmHg. A cortisol level of 1800 IU/litre excluded adrenal insufficiency. Urine testing remained positive for blood and the continuing absence of red blood cells on microscopy together with a serum creatine phosphokinase (CPK) greater than 10 000 IU/litre made rhabdomyolysis a likely cause.

Her cardiac function improved, such that inotropic support could be stopped, but as she was now oliguric, with no response to frusemide, and because of fluid retention and hyperkalaemia, peritoneal dialysis was commenced. Unfortunately, she soon developed a fever and peritonism probably due to yeasts (*Candida albicans*) which were isolated from sputum, skin swabs and dialysate fluid. The situation was further complicated by severe gastrointestinal bleeding. Blood, urine and cerebrospinal fluid cultures remained negative and a drug screen, although negative for salicylates and paracetamol, was positive for urinary phenothiazines.

Three weeks after her initial admission she was transferred to Hammersmith Hospital for haemodialysis. Phenytoin and diazepam had been given during the previous week for grand mal epileptic fits but had been stopped prior to transfer. Her trachea was intubated but she was breathing spontaneously. A chest X ray showed bilateral basal collapse, but despite appropriate antibiotics her temperature rose to a plateau of 42°C. As she was unable to maintain normocapnia, IPPV was resumed.

Neurological assessment showed that her conscious level varied between being awake and comatose. She had diminished reflexes together with extrapyramidal rigidity and a proximal weakness sparing the fingers and toes. A diagnosis of neuroleptic malignant syndrome was made and treatment was started with dantrolene sodium 20 mg intravenously three times daily. The creatine phosphokinase level had already fallen to less than 1000 IU/litre and the urine was now clear of blood although phenothiazines were still detectable.

She had been relatively stable during the 3 weeks since her transfer, but her condition now began to deteriorate. She had further gastrointestinal bleeding and endoscopy revealed two large duodenal ulcers. To avoid the use of heparin, haemodialysis was stopped and peritoneal dialysis restarted, but the dialysate was found to contain pus cells and yeasts and a laparotomy was performed to lavage the peritoneal cavity. Postoperatively, she required large blood transfusions to cope with continued gastrointestinal bleeding complicated by disseminated intravascular coagulation. A second laparotomy failed to halt the bleeding and she died soon after.

Discussion

Smego and Durack[2] found only 37 cases of the neuroleptic malignant syndrome in the English literature since its first report in 1968. It has been well investigated in France and Japan and more recently in Australia.[3] It is characterised by a slow onset, over a period of 24–72 hours, of generalised muscle hypertonicity, described as lead-pipe or plastic, together with akinesia. At the same time, or soon afterwards, the temperature rises to 41–42°C. The conscious level varies from alertness to confusion and mutism to coma, with associated sialorrhoea, dyskinesia and dysphagia. Autonomic dysfunction is manifested by tachycardia, labile blood pressure, diaphoresis and incontinence. Respiratory insufficiency is probably secondary to pneumonia, pulmonary emboli or aspiration due to dysphagia.[4]

Most cases follow treatment with haloperidol (a butyrophenone) or fluphenazine (a piperazine phenothiazine).[2] Other phenothiazines have been implicated, but it is more common with the longer-acting depot preparations. A case has been reported following the use of tetrabenazine and methyltyrosine (catecholamine depletors) in a patient with Huntington's disease.[5] More recently, a patient developed hyperthermia, muscle rigidity, acidosis and coma following an overdose of imipramine (a tricyclic antidepressant) and who was already taking tranylcypramine (a monoamine oxidase inhibitor).[6] Hyperthermia and muscle rigidity are recognised complications of treatment with monoamine oxidase inhibitors and tricyclic antidepressants, but it is interesting that this patient was taking Parstelin, a combination of tranylcypromine and trifluoroperazine. The latter is a long-acting piperazine phenothiazine with potent antidopaminergic activity and could therefore have been the cause of the patient's symptoms and signs. It is associated with therapeutic and not toxic doses, often at the start of treatment, change of dose, or the addition of a second drug.[7] A case thought to be exacerbated by hot weather has been reported[8] and dehydration and exhaustion are recognised contributory factors.[9]

The laboratory findings include a leucocytosis of 15–30 000 cells/cu mm, with or without a left shift. Liver function tests may show mild elevations of transaminases, lactate dehydrogenase and alkaline phosphatase, which may reflect fatty change in the liver induced by hyperpyrexia.[10] Creatine phosphokinase levels as high as 16 000 IU/litre and raised muscle aldolase may be seen, together with myoglobinuria due to rhabdomyolysis. The electroencephalogram, computerised axial tomography scans, cerebrospinal fluid and postmortem histology are usually normal.[11]

The condition affects all ages, but there is a male to female ratio of 2:1 and 80% of cases are under 40 years of age. Functional psychoses and, to a lesser extent, organic brain disease are said to be predisposing factors, but these patients are obviously at high risk of receiving neuroleptic drugs. In most cases, the neuroleptic syndrome is self-limiting, lasting 5–10 days after oral neuroleptics, but two to three times longer after depot preparations. The mortality is reported as 20–30%. It has been shown that the symptoms and signs resolve in parallel with the decrease in phenothiazine breakdown products in the urine.[12] The half-life of fluphenazine decanoate, the offending drug in the case reported, is longer than 3 weeks.[13]

In the past, schizophrenia as well as delerium tremens and insomnia have been listed as causes of rhabdomyolysis[14] and may well represent cases of neuroleptic malignant syndrome. Acute lethal catatonia, a condition reported in the literature for decades, is a syndrome of hyperactivity prior to stupor, hyperthermia and sometimes muscle rigidity. Only a few cases have been attributed to drugs;[15] the rest are diagnosed as schizophrenics or organic brain disease and may therefore have received neuroleptics. It can be difficult to distinguish from the neuroleptic malignant syndrome; the creatine phosphokinase is usually raised, the mortality is high[16] and they may, in fact, be the same entity.[17]

Central dopaminergic pathway blockade is a characteristic of the drugs implicated.[2] Raised levels of dopamine metabolites (3,4-dehydroxyphenylacetic acid and homovanillic acid) have been found at postmortem.[18] Dopaminergic pathways have been demonstrated in the hypothalamus and may involve the thermoregulatory centre, which would explain the hyperpyrexia seen. On this basis, dopaminergic drugs (L-dopa with carbidopa, bromocriptine and amantidine) have been used to treat neuroleptic malignant syndrome with variable success.[19–22] Dopamine has been shown to inhibit the contraction of skeletal muscle and this can be blocked by phenothiazines.[23]

Because of some similarities with malignant hyperthermia, dantrolene has also been used, in most cases with success.[24] It seems likely that the excessive heat production is a primary muscle phenomenon,[25] with or without an elevation of the hypothalamic thermoregulatory set point. Dantrolene reduces the amount of available intracellular calcium by blocking the calcium efflux in the muscle cell associated with the sarcolemmal action potential. It therefore interferes with the excitation–contraction coupling and acts as a direct-acting muscle relaxant. Indeed, it was first introduced for the treatment of spasticity.[26] It is also known to act on smooth muscle, cardiac muscle and the adrenal medulla and may act on other calcium dependent pathways. In doses as small as 100 mg/day it reduces the muscle rigidity, hyperpyrexia and the excessive oxygen consumption of this condition.[25]

Calcium is known to be important as a central nervous system regulator and membrane bound molecules called calcium dependent regulator proteins (CDRP) influence intracellular processes and their activity can be modulated by dopamine.[27] Individual idiosyncrasies of CDRP storage, release or deactivation may be responsible for the 'at risk patient'. Calmodulin, an intracellular protein found in all eukaryotic cells studied so far,[28] binds calcium and in doing so activates enzyme systems. Examples include adenylate cyclase, calcium and magnesium ATPase, phospholipase A_2, glycogen synthetase and myosin light chain kinase. It has also been implicated in neuromuscular transmission, platelet function, insulin release and smooth muscle contraction. It is inhibited by many drugs, including alpha-adrenoceptor blockers, antihistamines and local anaesthetics. Recently phenothiazines have been shown to be powerful calmodulin inhibitors.[29]

The link between malignant hyperthermia and neuroleptic malignant syndrome now looks tenuous.[30] Firstly, the triggering agents differ. Cases of neuroleptic malignant syndrome have undergone surgery without anaesthetic problems, including one patient who received suxamethonium on nine occasions for electroconvulsive therapy after recovering from neuroleptic malignant syndrome and who, 3 years earlier, at the age of 46 years, had undergone appendicectomy.[31] Known patients with malignant hyperthermia are not triggered by neuroleptics. Although muscle biopsies from neuroleptic malignant syndrome patients have been shown to produce abnormal contracture responses to phenothiazines and halothane,[32] the caffeine response is normal.[27,33] Biopsies from malignant hyperthermia patients respond normally to phenothiazines. Secondly, the slow onset, lower mortality and lack of family history also distinguish neuroleptic malignant syndrome from malignant hyperthermia. Unlike the latter, muscle enzymes are always normal after an attack.

Non-depolarising muscle relaxants have no effect on the rigidity seen in malignant hyperthermia, whereas they produce flaccid paralysis in all cases of neuroleptic malignant syndrome. Finally, there have been no reports of hyperthermia induced by neuroleptic drugs alone, in patients who are known to be susceptible to, or have a family history of, malignant hyperthermia.[5]

Although it seems likely that the symptoms and signs of neuroleptic malignant syndrome are central in origin and may be mediated by dopamine blockade or calcium or both, there also seems to be a primary muscle lesion. The site of this is unlikely to be the same as that in malignant hyperthermia and the two syndromes are probably distinct entities. In view of the widespread use of neuroleptic drugs outside of the confines of a general hospital, neuroleptic malignant syndrome may be far more common than current reports suggest.

We have presented a case which illustrates the potential complications and severity of this syndrome. The prophylactic use of dantrolene and dopaminergic drugs before surgery in at-risk patients, has been proposed by those in favour of a link between neuroleptic malignant syndrome and malignant hyperthermia.[34] Until more is known about the pathophysiology of neuroleptic malignant syndrome, it certainly seems prudent to use a combination of dantrolene and dopaminergic drugs in its treatment.

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CASE REPORT

Anaesthesia in the prune-belly syndrome

A review of 36 cases

A. M. HENDERSON, C. J. VALLIS AND E. SUMNER

Summary

Between 1959 and 1984, 36 children with the prune-belly syndrome underwent 133 operations at the Hospital for Sick Children, Great Ormond Street. The anaesthetic and surgical problems have been reviewed. Minor postoperative respiratory tract infections followed eight anaesthetics. Three deaths occurred in the postoperative period. In two of these there were multifactorial causes, not directly related to the prune-belly syndrome. One death was related to the sequelae of the syndrome. Normal doses of muscle relaxants are recommended when intermittent positive pressure ventilation is used during anaesthesia. Monitoring of the patient's respiratory state and active physiotherapy are advisable postoperatively. Analgesics should be used with caution.

Key words

Anaesthesia; paediatric, prune-belly syndrome.
Complications; pneumonia, uraemia.

The prune-belly syndrome (PBS) is characterised by the absence of muscle in the lower central and medial part of the abdominal wall, cryptorchidism and urinary tract abnormalities.[1–3] The skin of the anterior abdominal wall lies in redundant folds and its appearance in the newborn gives the syndrome its name (Fig. 1). Bilateral cryptorchidism and dilatation of the ureters and bladder are constant features.[4–10] Hydronephrosis with small dysplastic kidneys is commonly found. Abdominal muscle defects are seen in both sexes in a variety of rare conditions (for example, arthrogryposis and pterygium syndrome) but true PBS is confined to males.[11] The aetiology and true incidence of this rare condition are unknown. The chromosomes are usually considered to be normal; however, it has recently been suggested that the most likely abnormality is a two-step autosomal dominant mutation with sex-limited expression that partially mimics X-linkage.[12] There is no familial incidence.

All cases of the PBS have major anomalies in the urinary tract, but the severity of the condition varies considerably. In the most severe forms of the condition the babies are stillborn or die in the neonatal period, usually from uraemia or pulmonary hypoplasia. Some patients present

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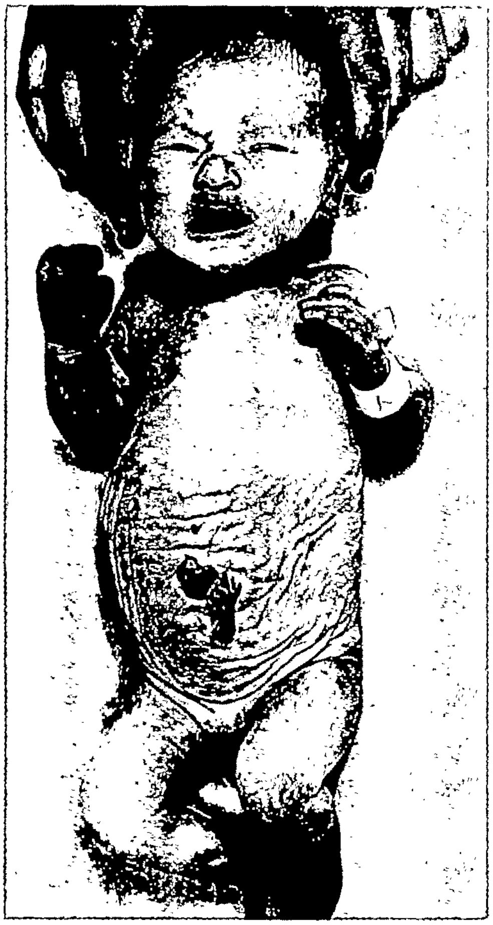

Fig. 1. The prune-belly syndrome.

with complete urethral obstruction. However, a large group have no symptoms relating to the urinary tract in spite of major abnormalities and do not usually require surgery. Many children with a patent urethra have such severe urinary tract abnormalities that they develop urinary tract infections with chronic renal failure and often require operations to improve drainage and prevent further renal damage. Reduction cystoplasty, ureteric reconstruction, nephroureterectomy and urinary diversion may be indicated in some cases. Plication of the abdominal wall is occasionally performed for cosmetic reasons. The testes are intra-abdominal and orchidopexy usually involves laparotomy.

The syndrome is associated with talipes equinovarus and congenital dislocation of the hips. Other associations include imperforate anus and malrotation of the gut. Children with PBS are particularly prone to recurrent urinary tract infections. Respiratory tract infections are also common, due to the children's inability to cough effectively and the retention of secretions.

Anaesthesia is thought to be hazardous in the PBS due to the absence of abdominal muscles, which could possibly lead to respiratory difficulty both intra- and postoperatively. There are, however, only two case reports of anaesthesia in the PBS, both of which support this view.[13,14] As many patients with the PBS are referred to the Hospital for Sick Children, we have been able to assess the risks associated with anaesthesia in a large series of patients.

Review of cases

The hospital's computerised record system was searched and the notes of all patients in whom PBS had been diagnosed during the period 1959–1984 were examined. Any case that had received an anaesthetic was admitted to the study. Only children with the true prune-belly syndrome were selected. Incomplete forms of the syndrome, such as those with a wrinkled prune-like skin, yet possessing some underlying muscle (pseudoprunes) and those with normal abdominal wall but dilated ureters and bladder, were excluded. The anaesthetic records, drug prescription charts, inpatient notes, X ray reports and discharge summaries were studied. Intra-abdominal and orthopaedic procedures were classified as major procedures. Investigative procedures such as cy-

Table 1. Associated abnormalities in 36 cases of prune-belly syndrome.

	Number
Talipes equinovarus	9
Congenital hip dislocation (CHD) and acrocephalic skull	1
Gross mental deficiency	1
Scoliosis	2
Scoliosis and plagiocephalic skull	1
Total	14

Table 2. Surgical procedures.

Major	
Abdominal genito-urinary	47
Abdominal non genito-urinary	44
Orthopaedic	10
Minor	
Investigations	20
Minor procedures	11
Adenotonsillectomy	1

Table 3. Age distribution of patients.

	Number	Percentage
Less than 1 month	8	6.7
1 month to 1 year	25	20.8
1 year to 5 years	48	40
5 years to 10 years	27	22.5
Over 10 years	12	10

stoscopies and limited surgical procedures, usually of the skin, were graded as minor.

Thirty-six cases of true PBS were reviewed. There were associated abnormalities in 14 cases (Table 1). A total of 133 anaesthetics were given and each patient received between one and thirteen. The operative procedures are shown in Table 2. Investigations included cystoscopy, cystogram, urethrogram, pyelogram, gastroscopy and sigmoidoscopy. Minor procedures included circumcision, catheterisation, urethral dilatation, urethrotomy and meatoplasty. The patients' ages ranged from 1 day to 16 years (Table 3). The majority of the patients were under 5 years of age and the respiratory system had not, therefore, yet reached maturity. Anaesthetic data were incomplete in 13 cases; data are presented on the remaining 120 anaesthetics. There was no recorded intra-operative morbidity or mortality in these cases.

All patients were judged pre-operatively to be fit for surgery. In all but three cases the conduct of anaesthesia, including premedication, followed the usual techniques chosen for all paediatric and neonatal cases anaesthetised in this hospital. Premedication consisted of atropine alone for infants under 1 year of age, compound injection of pethidine (pethidine 25 mg, chlorpromazine 6.25 mg and promethazine 6.25 mg/ml) 0.08–0.1 ml/kg, plus atropine for patients up to 15 kg or trimeprazine tartrate 2–4 mg/kg and atropine 0.05 mg/kg orally. Older children and those over 15 kg received papaveretum 0.3–0.4 mg/kg and hyoscine 0.08 mg/kg intramuscularly 90 minutes pre-operatively. Two patients, who presented before 1970, received rectal thiopentone.

Eight neonates (6.7%) were intubated awake. In 72 (60%) anaesthesia was induced with cyclopropane and oxygen, a technique still widely used in this hospital. Thirty-one patients (25.8%) were given thiopentone 4–6 mg/kg and nine (7.5%) were given an inhalation induction with nitrous oxide, oxygen and halothane. The choice of agent does not appear to have influenced anaesthetic morbidity. In all cases maintenance of anaesthesia involved nitrous oxide and oxygen. Halothane was added in 112 cases (84.2%). One patient was given ether (pre-1970). Intravenous analgesics were given to 14 patients. Ten received pethidine 0.3–0.5 mg/kg and four fentanyl 1.5–4 μg/kg.

Tracheal intubation was used in 93 patients (77.5%). This was facilitated by the use of suxamethonium in 82 patients and three were intubated following deep inhalational halothane anaesthesia. Eight neonates were intubated awake.

Sixty patients (50%) were allowed to breathe spontaneously through a modified Ayre's T-piece or Magill system (Mapleson A). Neonates and those having major procedures received artificial ventilation of their lungs. In 60 cases (50%) this was facilitated by the use of a muscle relaxant. Fifty-five of these patients received tubocurarine (mean dose 0.38 mg/kg, SD 0.15), one patient was given gallamine and four had intermittent suxamethonium. The neuromuscular block of all patients given tubocurarine or gallamine was reversed with neostigmine 0.05 mg/kg

Table 4. Anaesthetic data of patients who developed postoperative respiratory tract infections.

Patient	Weight (kg)	Age	Operation	Premedication	Induction	Suxamethonium	Halothane	Relaxant	Peroperative narcotic	Postoperative analgesics	No. of doses
9	28	10 years	Major	P 10 mg H 0.2 mg	Thio 4.2 mg/kg	+	+	TC 0.26 mg/kg	+	P 0.2 mg/kg	4
29	NA	2 years	Minor	Rectal thio A (? dose)	Cyclo/O_2	+	+	—	—	—	
34	10.7	15 months	Major	IPC 0.07 ml/kg	Cyclo/O_2	+	+	TC 0.3 mg/kg	—	CP	
23	13	10 months	Major	IPC 0.04 ml/kg	Cyclo/O_2	—	+	—	—	CP	2
18	9.5	11 months	Major	NA	Cyclo/O_2	—	+	TC 0.6 mg/kg	—	—	
16 (i)	9.4	16 months	Major	T 4 mg/kg A 0.3 mg/kg	Cyclo/O_2	+	+	TC 0.4 mg/kg	—	P 0.4 mg/kg	2
16 (ii)	18	5 years	Major	Peth 20 mg A 0.4 mg	Cyclo/O_2	+	NA	TC 0.25 mg/kg	—	—	
16 (iii)	21	6 years	Major	P 8 mg H 0.16 mg	Cyclo/O_2	+	+	TC 0.4 mg/kg	—	P 0.2 mg/kg	1

NA, not available; IPC, compound injection of pethidine; T, trimeprazine; A, atropine; TC, tubocurarine; Peth, pethidine; cyclo, cyclopropane; thio, thiopentone; P, papaveretum; CP, codeine phosphate (1 mg/kg); H, hyoscine.

and atropine 0.025 mg/kg at the end of the operation.

All patients remained in the recovery area until fully awake and the cardiovascular and respiratory states were stable. The patients were then returned to the ward. Postoperative analgesia was given when judged to be required by the attending nurse. Forty-four patients were given intramuscular injections of analgesics following major surgical procedures; 35 patients received papaveretum (mean dose 0.27 mg/kg, SD 0.05). Seven were given codeine phosphate (mean dose 1.05 mg/kg, SD 0.25) and two were given compound injections of pethidine 0.05 ml/kg.

Plasma urea was measured before 90 of the anaesthetics; 57 of these measurements were within the normal range of 3.0–7.0 mmol/litre. On 33 occasions, the plasma urea was elevated. Among the patients with 28 values between 7 and 17 mmol/litre, there were no deaths or major complications. Two patients had plasma urea values in the range 14–28 mmol/litre and one of these subsequently died. Two of three patients with a urea value above 28 mmol/litre died.

Serum potassium was measured pre-operatively on 90 occasions; 73 of these values were within the normal range of 3.0–4.8 mmol/litre. The value was elevated prior to anaesthesia for 18 urgent anaesthetics; 12 were between 5 and 6 mmol/litre, and 5 were between 6 and 7 mmol/litre. One patient's serum potassium was 7.2 mmol/litre but there were no documented dysrhythmias during the course of anaesthesia.

Postoperative respiratory tract infections followed eight of the 133 anaesthetics (Table 4). One patient had a respiratory tract infection after each of three procedures. On five occasions these patients required antibiotics in addition to physiotherapy. In all but one case the respiratory tract infection followed a major procedure. The type of anaesthetic these patients received did not differ significantly from those given to the rest of the group.

There were three deaths. One patient made an uneventful recovery from anaesthesia for an orthopaedic procedure for congenital dislocation of the hip. Twenty-four hours postoperatively he began to vomit repeatedly, possibly associated with uraemia (urea 34 mmol/litre). Aspiration of gastric contents was suspected since he developed severe bronchopneumonia and he died in spite of aggressive treatment. In the other two deaths, the cause was multifactorial and unrelated to the sequelae of the PBS. Both died in the neonatal period; one had multiple congenital abnormalities and a severe form of the PBS (urea 19 mmol/litre); the other was shown at post mortem to have fatty degeneration of the myocardium (urea 37.5 mmol/litre).

Discussion

The features of the PBS are well documented[1-6] and the surgical management has been extensively reported.[15-22] PBS is uncommon, but individual cases frequently require repeated surgical procedures and may occasionally be encountered outside specialist paediatric centres. The descriptions of anaesthetic management, however, are confined to only two case reports.[13,14]

Hannington-Kiff[13] reported anaesthetic difficulties in a 3-year-old child with PBS. The patient developed severe, nonobstructive, paradoxical respiration during anaesthesia which was treated successfully with assisted ventilation. Karamanian *et al.*[14] described a case of PBS in which postoperative respiratory failure developed and required positive pressure ventilation. This child had a previous history of recurrent respiratory tract infections and underwent a very long operation. Both papers emphasise that anaesthesia in PBS is hazardous due to the absence of the abdominal muscles, which may lead to respiratory difficulties.

We report the results of anaesthesia in PBS administered by many anaesthetists of varying degrees of seniority. The anaesthetic techniques employed, apart from three cases early in the series, were essentially the same as those used for similar procedures in non-PBS patients in this hospital. In a retrospective analysis such as this, a complete picture cannot be obtained of the incidence of complications which arise during the course of anaesthesia. However, it is unlikely that any major complications, such as severe respiratory difficulties during anaesthesia or any serious postoperative problems, would have gone unrecorded. In our series there were no reports of any intra-operative respiratory difficulties even in the 60 anaesthetics where patients were allowed to breathe spontaneously for minor surgery.

Although postoperative respiratory tract infections followed eight of the 133 anaesthetics, it should be noted that these were all relatively mild and resolved completely with antibiotics and physiotherapy. No patient required postoperative respiratory support.

Three deaths occurred in the postoperative period. Uraemia was present in all three cases. In two of these there were multifactorial causes in addition to the sequelae of the PBS. One death was directly related to the sequelae of the PBS. This patient was uraemic and developed pneumonia secondary to repeated vomiting. The absence of abdominal muscle in this child was probably a contributory factor.

From our experience, we recommend that general anaesthesia in PBS should be undertaken with particular care in view of the *potential* risk of respiratory difficulties in the postoperative period. Careful pre-operative assessment is advisable in all cases. Any existing respiratory infection should be thoroughly treated before commencing elective surgery. Patients with uraemia may have an increased risk of vomiting at induction and in the postoperative period; although none of our patients vomited at induction many did in the immediate postoperative period. Moderate doses of premedicant drugs were well tolerated. Large doses should be avoided, due to the risk of respiratory depression. Intermittent positive pressure ventilation combined with neuromuscular blockade was employed in half the anaesthetics. Atracurium may be the muscle relaxant of choice in the patients with known renal impairment. The other patients were allowed to breathe spontaneously for minor surgery and this appears to be a safe technique.

Active physiotherapy is recommended and, ideally, should be commenced pre-operatively to familiarise the child with the techniques. Close observation is necessary postoperatively and the continued need for physiotherapy should be assessed.

Caution should be exercised in the use of postoperative analgesics known to produce respiratory depression, but it is not necessary to withhold them.

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Anaesthesia, 1987, Volume 42, pages 61–66

CASE REPORT

Insertion of intratracheal stents

Anaesthetic management using high frequency jet ventilation or cardiopulmonary bypass

K. M. SHERRY, P. A. KEELING, H. M. JONES AND W. AVELING

Summary

The case histories of two patients who required the insertion of intratracheal stents are presented. The peroperative anaesthetic management of one case included the use of high frequency jet ventilation of the lungs; in the other case, partial cardiopulmonary bypass was used. The advantages and disadvantages of these two methods are discussed.

Key words

Surgery; tracheal.
Ventilation; high frequency jet.
Technique; cardiopulmonary bypass.

An intratracheal stent is a silicon rubber prosthesis designed for use in the management of difficult tracheal stenosis. Tracheal stents were first used in 1968[1] and have, since then, been inserted either for the long-term management of tracheal collapse or for palliation of malignant compression prior to radiotherapy treatment. There are two types: a simple T-piece (Montgomery) for lesions in the proximal two-thirds of the trachea and a bifurcating prosthesis (Westaby)[2] for compressions of the distal trachea and proximal bronchi.

The surgical procedure for tracheal stent insertion under general anaesthesia involves initial bronchoscopy to assess the extent of the stenosis, to determine the length of the trachea and to establish the site for the tracheostomy incision. The trachea is then opened through such an incision. Two fine gum elastic bougies are placed through the tracheostomy and the trachea and into the two main bronchi under direct vision, for the insertion of a bifurcating prosthesis. The stent is railroaded into position over the guides with the T-limb protruding through the tracheostomy stoma. The upper intratracheal portion of the stent is then manipulated to lie within the subglottic trachea.[2] A single intratracheal guide is used for the simple T-piece prosthesis (Fig. 1).

During previous insertions of these stents at this hospital, ventilation of the lungs has been maintained manually, using the Sanders attachment for bronchoscopes.[3] However, this has not always provided adequate oxygenation throughout the surgery and has hindered access to the surgical field. Our experiences led us to use high frequency jet ventilation of the lungs for peroperative oxygenation and postoperative respiratory support.

These case reports illustrate the use of high frequency jet ventilation of the lungs and partial cardiopulmonary bypass in the anaesthetic

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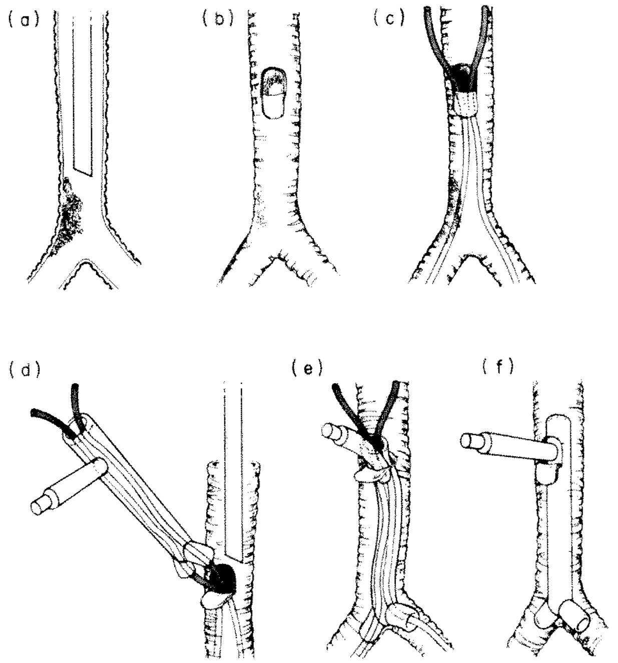

Fig. 1. Diagram to show the surgical procedure for the insertion of a bifurcating intratracheal stent. (By kind permission of Mr S. Westaby.) (a) Initial bronchoscopy; (b) opening of the tracheostomy; (c) bougies positioned in the main bronchi under bronchoscopic guidance; (d) and (e) railroading the stent into position; (f) final position of the stent within the trachea.

management of patients for insertion of these stents.

Case histories

Case 1

A 58-year-old female presented for surgery with tracheal compression secondary to a retrosternal recurrence from a thyroid tumour. Eighteen months prior to admission she had had a total thyroidectomy with block dissection of the left cervical lymph nodes; one month prior to admission she developed symptoms of tracheal compression. Fibre-optic bronchoscopy 2 days prior to surgery demonstrated collapse of the intrathoracic trachea and distorted slit-like bronchial openings. Laser resection of the tumour through a bronchoscope was attempted, but failed to provide symptomatic improvement.

At the time of pre-operative assessment she was distressed and unable to lie flat. The blood pressure was 180/70 mmHg and heart rate was 140 beats/minute and irregular. She had inspiratory and expiratory stridor, coarse crepitations in both lung fields and venous engorgement of the head and neck. Pre-operative haemoglobin and plasma urea values were normal, and serum sodium was 130 mmol/litre and serum potassium was 3.3 mmol/litre. Chest X ray showed a stenosis of the distal two-thirds of the trachea and bilateral bronchopneumonia secondary to inhalation of blood and debris during the laser resection. An electrocardiogram showed that the cardiac rhythm varied between atrial fibrillation and sinus tachycardia with supraventricular ectopics. Pre-operative arterial blood gas tensions are shown in Table 1.

No pre-operative premedication was prescribed. Intra-arterial blood pressure and monitoring of the electrocardiograph were established pre-operatively. Anaesthesia was induced with midazolam 5 mg, oxygen and halothane. Manual ventilation of the lungs using a Magill system and mask was possible. Anaesthesia was subsequently maintained by incremental fentanyl to a total dose of 200 μg. A continuous infusion of etomidate 0.15 mg/kg/minute was started and the patient was paralysed using vecuronium to a total dose of 12 mg. Initial bronchoscopy was performed using a small Negus bronchoscope.

Table 1. Case 1. Arterial blood gas tensions.

	Before induction	Sanders attachment 16-G needle	HFJV 16-G needle	HFJV 9-G cannula	Postoperatively
$F\mathrm{IO}_2$	0.35	1.0	1.0	1.0	0.5
$P\mathrm{aO}_2$ (kPa)	9.5	13.1	8.5	12.1	19.6
$P\mathrm{aCO}_2$ (kPa)	4.63	5.92	4.53	5.96	5.9

Pre-operative measurement immediately before induction of anaesthesia; HFJV, high frequency jet ventilation. Postoperative measurement 9 hours after weaning from high frequency ventilation, 18 hours after operation.

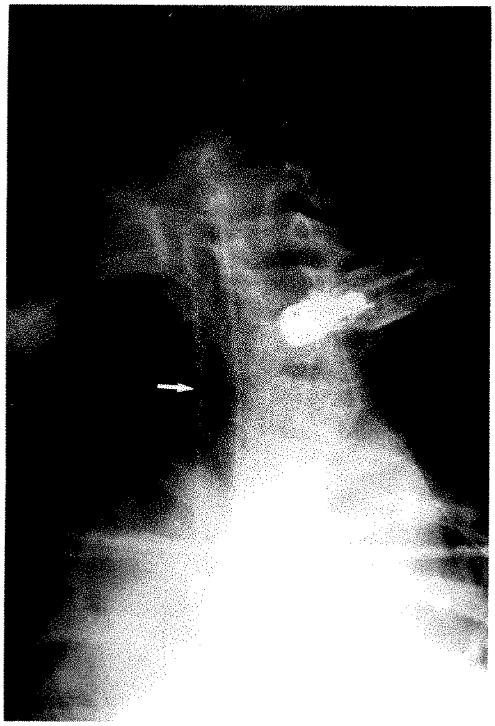

Fig. 2. Case 1. Postoperative chest X ray to show the bifurcating tracheal stent in position.

Ventilation of the lungs was first established by the use of the Sanders ventilating attachment for bronchoscopes, with a 16-G needle, and then changed to the Acutronic MK800 ventilator connected to the same needle and set to deliver 100 breaths/minute. The bronchoscope was left within the trachea and high frequency jet ventilation of the lungs was continued during the tracheostomy and placement of the guides into the two main bronchi. At this time ventilation was discontinued, the bronchoscope was withdrawn until it just protruded through the glottis and the stent was railroaded into position over the guides. High frequency jet ventilation of the lungs was then recommenced through a flexible 9FG feeding catheter (Pennine), introduced through the T-piece and into the tracheal portion of the stent. The operative procedure lasted for one hour. Postoperative ventilatory support was required and high frequency jet ventilation was delivered to the lungs via the same catheter through the tracheostomy. The patient was weaned from ventilatory support over the ensuing 9 hours. On the following morning the T-piece of the stent was closed with a spigot; the patient had no stridor and could cough and speak normally. She remained well symptomatically until the third postoperative day when she suffered a fatal pulmonary embolus. Per- and postoperative arterial blood gas tensions are shown in Table 1 and the postoperative chest X ray with the stent in place is shown in Fig. 2.

Case 2

A 22-year-old-female presented with a 7-months' history of increasing breathlessness, stridor and chest pain. She had an 8-year history of wheezing, diagnosed as asthma and treated by inhaled steroids and salbutamol. She also had a strong family history of atopy.

On admission her chest X rays and tracheal tomograms showed near occlusive narrowing of the trachea, extending from the subglottic region to within one centimetre of the carina (Fig. 3). Bronchoscopy and tracheal biopsy established a histological diagnosis of a slow-growing tracheal cylindroma. Lung function tests were typical of a severe obstructive defect: forced vital capacity (FVC) was 2590 ml, forced expiratory volume in 1 second (FEV_1), 1190 ml and FEV_1/FVC, 0.46.

She developed severe respiratory distress necessitating relief of respiratory obstruction within 24 hours of the initial radiotherapy. Bronchoscopic resection of her tumour and the insertion of a bifurcating tracheobronchial stent

Table 2. Case 2. Arterial blood gas tensions.

	Before induction	Bypass and injector	Bypass alone	Bypass and injector via stent
F_{IO_2}	0.4	1.0	0	1.0
Pa_{O_2} (kPa)	35.9	16.1	9.2	33.5
Pa_{CO_2} (kPa)	3.87	5.87	5.73	3.87

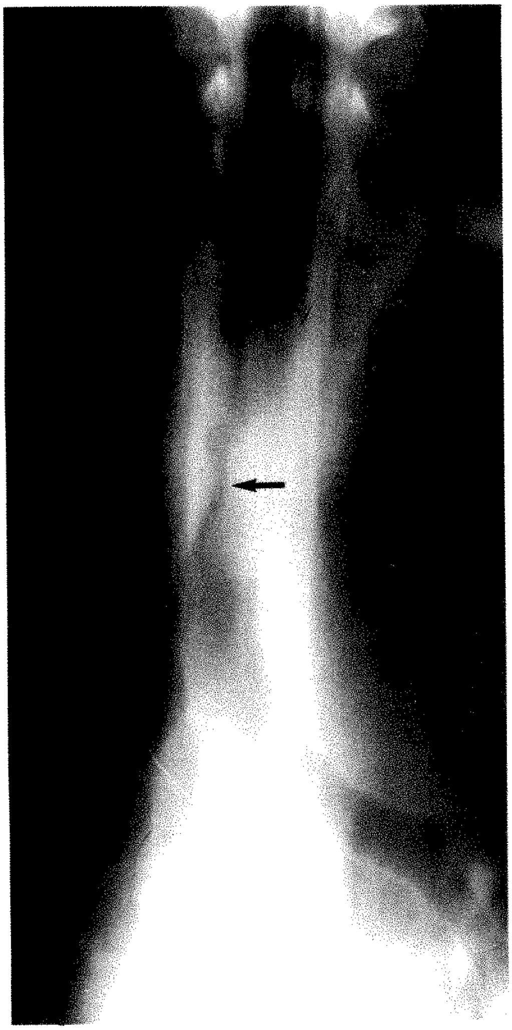

Fig. 3. Case 2. Pre-operative chest X ray to show the tracheal stenosis.

were planned. It was decided that peroperative oxygenation should be provided by a combination of ventilation of the lungs and elective partial femorofemoral bypass because of the possibility of peroperative respiratory obstruction.

No pre-operative premedication was prescribed. Intra-arterial pressure, central venous pressure and electrocardiographic monitoring were established pre-operatively. Arterial blood gas tensions were measured (Table 2). The patient was sedated with midazolam 5 mg and fentanyl 100 μg intravenously and anticoagulated with heparin 3 mg/kg. The femoral artery and vein were exposed and cannulated under local anaesthesia. General anaesthesia was induced with etomidate 0.3 mg/kg while the patient breathed 100% oxygen, and was maintained with an etomidate infusion 0.1–0.2 mg/kg/minute. Partial extracorporeal circulation was established and maximum flows of 2 litres/minute were obtained. When partial cardiopulmonary bypass had been established the patient was cooled to 32°C and the blood pressure fell from 140/80 mmHg to 80/60 mmHg. The heart remained in sinus rhythm and continued to eject.

A Negus bronchoscope was passed and the lungs were ventilated using the Sanders venturi injector via a 16-G cannula. While the tumour was being resected the trachea became completely obstructed by blood and debris for 8 minutes before the stent was inserted. During this time, low but adequate arterial oxygenation was maintained by the partial cardiopulmonary bypass perfusion. Following insertion of the stent, ventilation of the lungs was again established using the Sanders venturi injector and the 16-G needle through the T-piece of the prosthesis. The extracorporeal perfusion was withdrawn and anticoagulation was reversed using protamine sulphate after rewarming the patient to 37°C.

Postoperative respiratory support was provided using the Acutronic MK800 high frequency ventilator via a catheter through the tracheal T-piece. The ventilator was set to deliver 150–200 breaths/minute, with an inspired oxygen concentration of 50%. This maintained a Pa_{CO_2} of 3.9–4.8 kPa with Pa_{O_2} greater than 13 kPa (Table 2). The patient was weaned from ventilatory support overnight. Pre- and peroperative lung function tests were FVC 3800 ml, FEV_1 1800 ml and FEV_1/FVC 0.47, and her stridor was relieved.

The patient continued her 6-week course of radiotherapy without further deterioration in the patency of her airway. However, radiotherapy failed to arrest the progression of her cylindroma and she died 5 months later.

Discussion

Surgery or radiotherapy in patients with tracheal stenosis may lead to a deterioration of the airway due to an increase in tissue oedema. Traditional therapies are aimed at reducing the symptoms by humidifying the inspired air or by using oxygen in helium as the inspired gas, and at reducing the oedema by the use of topical vasoconstrictors and systemic steroids. Surgery is indicated for patients in whom the patency of the trachea is in jeopardy despite medical treatment. When the lesion is intrathoracic a conventional tracheostomy is rarely effective because the block is too low; however, the use of a tracheal stent can bypass the obstruction. The stent also enables the patient to breathe normally through the upper airway and to humidify his own respiratory gases whilst retaining the ability to speak and to clear secretions. The tracheal T-piece of the stent enables supplementary suction of the tracheobronchial tree when this is required. Furthermore, the same tube may be left in position for many months without adverse effects.

High frequency jet ventilation has an established clinical role in the management of patients for laryngoscopy,[4] bronchoscopy[5] and tracheal surgery.[6-8] It has been shown to provide good oxygenation and carbon dioxide elimination[9,10] at low peak airway pressures[11] and to prevent aspiration of blood and secretions into the lungs.[12] The use of partial femorofemoral bypass has been previously described during the surgical management of severe airway obstruction.[13,14]

The decision on the method of peroperative oxygenation in these two patients was determined following discussion with the surgeon. In case 1 the stent was to be inserted as a palliative procedure without attempting resection, whereas in case 2 the vascular tumour was to be debulked surgically prior to insertion of the stent. Tumour resection greatly increases the likelihood of obstruction of the large airways from bleeding and tumour debris, and poor gas exchange may result from aspiration of blood into the lungs. These were the major factors in determining whether prophylactic partial femorofemoral bypass was indicated.

The use of muscle relaxants in patients with respiratory obstruction is controversial. Tumours which do not compromise the airway when the patient is awake may precipitate total respiratory obstruction when muscle tone is lost. This is more likely to occur in association with tumours of the larynx, where maintenance of the airway is more dependent on skeletal muscle tone, than in tracheal lesions, where support of the lumen is from the fibrous wall and cartilaginous rings. However, in patients with tracheal and bronchial tumours, intermittent positive pressure ventilation of the lungs may precipitate a ball valve type of obstruction. For these reasons a patient with respiratory obstruction should never be paralysed until it has been established that controlled ventilation is possible with a facemask. In case 1, muscle relaxation was indicated in order to facilitate the surgery and a relaxant was given after it had been shown that manual ventilation of the lungs was possible. In case 2 it was possible to ventilate the lungs and carry out the surgery during partial cardiopulmonary bypass with cooling and sedation and without muscle relaxants.

In case 1, high frequency jet ventilation of the lungs was provided using the Acutronic MK800 ventilator, initially via a 16-G needle attachment to the bronchoscope and then via a Pennine 9FG catheter. The Pennine catheter, which is 38 cm long, was mounted in a T-piece adaptor on the tracheostomy limb of the prosthesis at 12 cm from its distal end and opened into the tracheal portion of the stent. Ventilation was monitored peroperatively by auscultation of the chest and by repeated arterial blood gas analysis. Bilateral auscultation confirmed the correct placement of the catheter; however, in future, bilateral stethoscopes strapped to the chest wall would enable continuous monitoring of air entry to both lungs. There is a period of obligatory apnoea during the insertion of the stent. However, if the guide wires were cannulated, continuous ventilation could be delivered to both main bronchi independently throughout surgery. The use of high frequency jet ventilation in the management of this patient had advantages over our previous practice of manual ventilation of the lungs using the Sanders attachment. Continuous ventilation of the lungs was maintained throughout the operation, with the exception of the short interval while the stent was railroaded into position, gas exchange was good and the hands of the anaesthetist were freed for other aspects of the anaesthesia. It also facilitated the comfortable, easy weaning of postoperative respiratory support and avoided the necessity to resort to tracheal intubation. The potential disadvantage of high frequency jet ventilation is an inability to provide ventilation to the lungs, either because a catheter cannot be passed beyond the obstruction or because the airway is narrow and air trapping occurs within the lung.

In the second case, the risk of peroperative total occlusion of the airway was high and in this circumstance cardiopulmonary bypass offers the only certain way to maintain oxygenation. The advantages of partial femorofemoral cardiopulmonary bypass are that it can be relatively easily established under local anaesthesia and it ensures adequate arterial blood gas tensions. In case 2, although there was a decrease in arterial blood gas tension during the period of total respiratory obstruction because full bypass was not established and the heart continued to eject, oxygenation was adequate. Partial cardiopulmonary bypass provides some hypotension which reduces surgical bleeding and allows moderate hypothermia as a means of cerebral protection. The drawbacks of this technique are that it is only available in hospitals with facilities for cardiac surgery and that it is invasive. Heparinisation may increase the surgical bleeding, but this was not a problem during this case and the surgeon was able to resect sufficient tumour for the stent to be inserted.

In summary, near occlusive lesions of the trachea and their surgical management are a major challenge to the skill of the anaesthetist and surgeon. The use of high frequency jet ventilation of the lungs proved useful in the anaesthetic management of these cases undergoing intratracheal stent insertion. However, when surgical resection of the tumour is indicated, as in case 2, extracorporeal perfusion may be the only method of ensuring oxygenation.

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APPARATUS

Flow through intravenous cannulae

I. G. KESTIN

Summary

The flows through intravenous cannulae, generated using the test described in British Standard 4843, are presented for all the cannulae available in the United Kingdom. These flows are compared to those obtained using clinical apparatus with two different pressure gradients. The implications of these results in selecting suitable intravenous cannulae and the other factors influencing flows through cannulae are discussed.

Key words

Equipment; catheters, venous.

In 1972 the British Standards Institution published a standard governing the manufacture of peripheral venous cannulae, BS4843.[1] In this standard, a test for determining the flow through cannulae is described and this information has to be included on the packaging of every intravenous cannula in order to conform with the requirements of BS4843. This information provides the clinician with some guidance in choosing a suitable gauge cannula.

This test has not been universally adopted by the manufacturers and even if the information is known, it is often not included on the packaging. This study compares the results obtained using the conditions specified in BS4843 with flows obtained using clinical apparatus.

Methods

Flows using the BS4843 test

The essential feature of the BS4843 test to determine the flow through a cannula is that a constant-level tank is used to provide 10 kPa pressure of distilled or de-ionised water at 22°C (SD 2°C) via 4-mm internal bore tubing to the cannula. The length of the connecting tubing must be 110 cm, with a male Luer fitting to the cannula. The terminal 4 cm of the tubing and the cannula are kept horizontal to ensure a streamline flow. The collection is made over at least 30 seconds and the results are expressed as the mean of three such determinations in ml/minute and must be accurate to within 10%.

Flows using clinical apparatus

The flows obtained using the BS4843 test were compared with those using clinical equipment under two conditions. Instead of using a constant-pressure head water tank as in BS4843, a 500-ml bag of 0.9% saline (Travenol) was connected to a Travenol blood administration set and the junction of the giving set and bag placed 100 cm above the tip of the cannula. The ter-

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minal 4 cm of the administration set and the cannula were held horizontal as in the BS4843 test. The room temperature was 24°C and the flows were collected over 30 seconds. Each test was done using a full 500-ml bag of 0.9% saline and the mean of three consecutive readings is given.

Anaesthetists commonly infuse fluids under pressure and a similar test was therefore performed using a 1000-ml bag of 0.9% saline (Travenol) pressurised to 300 mmHg and using a previously calibrated pressure infusor. The saline was connected to the cannula by a Travenol blood administration set. The 1-litre bag of saline, giving set and cannula were kept in the same horizontal plane. The room temperature was 24°C, the collections were made over 15 seconds and the pressure was maintained at 300 mmHg for the duration of the collections. Each test was performed using a full 1-litre bag of 0.9% saline and the mean of three consecutive readings was taken.

Results

Table 1 shows the flows of the commonly used gauges and Table 2 gives the flows of all gauges of cannulae available in the UK. These data were obtained from the manufacturers and are complete except for the new Insyte cannula, which has not yet been tested.

Table 1. Flows in ml/minute, using the test conditions specified in BS4843, for the gauges in common use. If several length cannulae are available at the same gauge, the flow for the shorter length is given.

	14-G	16-G	18-G	20-G	22-G	24-G
Abbocath	267	137	81	42	26	16
Angiocath	301	148	82	49	27	18
Cathlon	360	220	120	80	—	—
Intraflux	266	139	97	50	23	—
Intravalve	264	141	100	53	23	—
Jelco	360	220	120	80	42	24
Medicut	306	225	114	63	27	—
Mediport	280	135	75	40	25	—
Mosquito	270	168	85	58	25	15
Quickcath	253	125	76	44	26	16
Venflon/ Vasculon	270	180	80	54	31	13 (Neoflon)
Wallace	251	121	48 (18.5-G)	39	25	18

Table 2. Flows using the BS4843 test for all gauges available in the UK.

Gauge	Mean flow (SD), ml/minute	Maximum flow, ml/minute	Minimum flow, ml/minute
10	577 (Angiocath)		
12	465 (59)	516 (Medicut)	400 (Mosquito)
13	348 (31)	370 (Mosquito)	326 (Wallace)
14	286 (35)	360 (Jelco, Cathlon)	251 (Wallace)
16	162 (35)	222 (Jelco, Cathlon)	121 (Wallace)
17	125 (Venflon)		
17.5	65 (Wallace)		
18	91 (17)	120 (Jelco, Cathlon)	73 (Wallace Y-can)
18.5	48 (Wallace)		
19	44.5 (Wallace Y-can, 31 via side channel)		
20	54 (14)	80 (Jelco, Cathlon)	33 (Abbocath)
21	26.5 (Wallace Y-can, 21.5 via side channel)		
22	28 (5)	42 (Jelco)	23 (Intravalve)
23	19 (Wallace Y-can, 16 via side channel)		
24	17 (3)	24 (Jelco)	13 (Neoflon)
26	9 (Abbocath)		

Table 3. Flows in ml/minute, via a blood administration set of 0.9% saline 100 cm above cannula.

	Flow (ml/minute)
14-gauge cannulae	
Abbocath	236
Angiocath	234
Cathlon	288
Intraflux	236
Intravalve	234
Jelco	280
Medicut	284
Mediport	236
Mosquito	234
Quickcath	230
Vasculon	234
Venflon	226
Wallace	230
10-gauge cannula	
Angiocath	386
Giving set alone	
Travenol blood administration set	408

The results of the flows obtained using the unpressurised bag of saline are shown in Table 3. The flows through a 10-gauge Angiocath, the largest peripheral venous cannula available, and through the administration set alone are included for comparison. The results obtained using the pressurised bag are shown in Table 4.

Table 5. Flows through the common gauge cannulae at which laminar flow will become turbulent using distilled water at 20°C. Calculated from Reynold's number $Re = 4\dot{Q}/\pi D\nu$, where Re = Reynold's number; $\dot{Q}$ = volume flow rate; D = diameter of tubing; ν = kinematic viscosity = viscosity/density = 1.0038 sq cm/second for distilled water at 20 °C. Re is taken as 2000 for turbulent flow.

Gauge	Internal diameter (mm)	Flow (litres/minute)
12	2.2	20.8
14	1.6	15.1
16	1.2	11.4
18	0.9	8.5
20	0.7	6.6
22	0.6	5.7
24	0.4	3.7
4-mm bore tubing	4.0	37.9

Discussion

The flows between cannulae of the same gauge but different manufacture vary by well over 10% (Table 1). There are variations in the internal diameter of cannulae of the same gauge between manufacturers, which will account for the major differences in flows shown in Table 1. Table 5 shows the flows through the cannulae at which turbulent flow occurs in the cannula itself, and clearly these are above the attainable flows. Nevertheless, different conformations at the hub–cannula junction may induce turbulence in some cannulae but not in others and may also account for some of these differences.

The blood administration set has an internal bore of 4 mm, as does the tubing specified in BS4843, but is of greater length and the addition of the filtration and drip chamber will impede flow compared to the BS4843 test conditions. This is demonstrated by comparing Tables 1 and 3,

Table 4. Flows in ml/minute, via a blood administration set of 0.9% saline under 300 mmHg pressure.

	14-G	13-G	12-G	10-G
Abbocath	496			
Angiocath	484		750	832
Cathlon	540			
Intraflux	456	600	732	
Intravalve	480			
Jelco	546			
Medicut	576		744	
Mediport	448			
Mosquito	496	580	720	
Quickcath	520			
Vasculon	444			
Venflon	480			
Wallace	472	570		
Mean	495	583	736	832

which show the decreased flows obtained if a giving set is substituted for a uniform-calibre tubing, despite the pressure gradient across the giving set to cannula tip being slightly more than the 100 cm used in the BS4843 test. Table 3 shows that the flow through a giving set alone is only marginally increased as compared to when a 10-gauge cannula is attached, so a 10-gauge represents the limit of cannula size using the current 4-mm internal bore blood administration set.

Infusing fluids through an administration set under pressure leads to only moderate increase in flows. The test described earlier using 0.9% saline pressurised to 300 mmHg gives an approximate fourfold increase in pressure gradient down the administration set and cannulae compared to the previous test or that of BS4843. However, comparing Tables 3 and 4, flows are only increased by between 91% and 126% according to gauge. This approximate doubling of flows with quadrupling of pressure gradient suggests that the overall flow pattern down an administration set and 14-gauge cannula is turbulent rather than laminar. At these flows, Table 5 shows that flow within the actual cannula and the tubing itself should be laminar so turbulence would only occur at the drip chamber and the cannula–hub–administration set junctions. When rapid transfusion is required, it is more efficacious to insert a large cannula than rely on pressurising the infusate through a smaller cannula.

Tables 1, 3 and 4 show that the Cathlon, Jelco and Medicut have flows which are considerably higher than the mean for the gauge. The 22-gauge Jelco has a greater flow than some 20-gauge cannulae. The Medicut is the only tapered cannula available and since the gauge is specified by the tip diameter, most of the cannula length will be greater than this diameter. This tapered conformation down to the narrowest part of the cannula without any abrupt changes in the internal bore, should encourage laminar flow.

The Jelco and Cathlon cannulae are specifically marketed as thin-walled cannulae and the introducer needle from a Jelco or Cathlon is too large to pass down other cannulae of the same gauge, except the tapered Medicut. The only potential disadvantage of thin-walled cannulae is that they may be more prone to kinking in use.

Many other factors influence the flow of an infusion in clinical practice apart from the cannula diameter. The flow using blood is considerably less than using water. At room temperature, flow is decreased to 50–60% of water flow.[2] The viscosity of blood rises sharply with haematocrit and decreasing temperature. Knight[3] showed that warming blood to body temperature doubles the flow compared to blood transfusion at 10°C.

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Appendix

List of manufacturers

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2. Argyle (Division of Sherwood Medical Industries, Ltd) (Medicut, Mediport), London Road, County Oak, Crawley, West Sussex RH10 2TL.
3. Critikon Ltd (Cathlon, Jelco), Broadlands, Sunninghall, Ascot, Berkshire SL5 9JN.
4. Deseret Medical (Angiocath, Insyte), Milton Trading Estate, Abingdon, Oxfordshire OX14 4RX.
5. Travenol Laboratories Ltd (Quickcath), Caxton Way, Thetford, Norfolk IP24 3SE.
6. Viggo (Vasculon, Venflon, Neoflon), British Viggo, Faraday Road, Dorcan, Swindon SN3 5HZ.
7. Vygon (UK) Ltd (Intraflux, Intravalve, Mosquito), Bridge Road, Cirencester, Gloucestershire GL7 1PT.
8. Wallace (Wallace, Wallace Y-can), H. G. Wallace Ltd, Commerce Way, Colchester, Essex CO2 8HH.

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Forum

Beta-adrenoceptor blockade and suxamethonium-induced rise in plasma potassium

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Summary

The effects of beta-adrenergic blockade on the suxamethonium-induced rise in arterial plasma potassium were studied in patients who presented for open heart surgery. No potentiation of the immediate rise in plasma potassium was observed.

Key words

Complications; hyperkalaemia.
Neuromuscular relaxants; suxamethonium.

There is increasing evidence to suggest that, by promoting the entry of potassium into the cells via beta$_2$-adrenoceptor stimulation of sodium, potassium-ATPase,[1] the sympatho-adrenal system may have an important influence on potassium homeostasis.[2–4] Thus, beta-blockade with propranolol has been shown to be associated with raised plasma potassium levels following long-term treatment in hypertensive subjects[5] and during cardiopulmonary bypass,[6] while exaggerated rises in plasma potassium levels have been reported during experimental exogenous potassium loading[7] and exercise.[8,9] The effect may be considerable, as is the case during exercise, where a peak arterial plasma potassium level of 7.8 mmol/litre was recorded 3 minutes after the start of exercise in a beta-blocked subject, compared with rises to 5.4 mmol/litre in non beta-blocked subjects.

This study was performed to investigate the possibility that beta-blockade may also affect the magnitude of the plasma potassium rise which is known to occur following the administration of suxamethonium.[10,11]

Method

Eighteen patients aged between 39 and 67 years, undergoing either coronary artery bypass grafting or aortic/mitral valve replacement, were studied. All patients were premedicated with lorazepam 1–2 mg, papaveretum 15–20 mg and hyoscine 0.3–0.4 mg. On arrival in the anaesthetic room, a radial artery cannula (Abbocath 18–20G) was inserted under local analgesia (lignocaine 2%) and a control arterial blood sample was collected for potassium and blood gas analysis. Anaesthesia was then induced with thiopentone 5–6 mg/kg injected intravenously over a 1-minute period via a 23-G butterfly needle. Another arterial blood sample was taken, following which suxamethonium 1.5 mg/kg was administered. Tracheal intubation was performed approximately 1 minute after this and then intermittent positive pressure ventilation with 67% nitrous oxide in oxygen was commenced. Further arterial blood samples were collected at 1, 2, 4 and 6 minutes after the administration of suxamethonium.

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All the samples were collected by withdrawing 1.5 ml blood into 2-ml heparinised syringes (1000 IU/ml heparin occupying the dead space in the nozzle of the syringe).

Plasma potassium was measured with an IL 501 sodium–potassium analyser. The performance of this machine had earlier been tested by analysing 20 blood samples (potassium range 1.7–13.8 mmol/litre) on this machine and an IL 342 flame photometer in the pathology laboratory. Comparison of these two methods yielded a regression line of $y = 0.96x + 0.18$ with an r value of 0.998. The coefficient of variation obtained from 18 repeated analyses on the same sample was 1.05%. Blood gas analysis was performed on an ABL 30 Acid Base Analyser.

Results

The patients were divided, for the purpose of analysis, into three groups on the basis of their pre-operative medication: group 1, diuretics or no treatment; group 2, propranolol 80–320 mg/day; group 3, cardioselective beta-blockers (atenolol/metoprolol/acebutolol). The measured plasma potassium levels for all the patients are shown in Table 1, while the mean changes in the three groups are shown in Table 2 and Fig. 1. The general pattern was for plasma potassium to decrease by approximately 0.1–0.2 mmol/litre during the induction with thiopentone, while administration of suxamethonium was followed by a small rise, detectable within 1–2 minutes of the injection and maintained until the end of the observation period (6 minutes after injection of suxamethonium). There were no significant differences in the plasma potassium changes between the three groups of patients. Mean $P\mathrm{aco}_2$ levels during the period studied are shown in Table 3. It will be seen that although $P\mathrm{aco}_2$ rose slightly during the induction–intubation sequence and then fell as intermittent positive pressure ventilation was instituted, there were no significant differences in the changes between the three groups.

Table 1. Measured plasma potassium levels (mmol/litre) in individual patients in group 1 (no treatment with beta-blockers), group 2 (treatment with propranolol) and group 3 (treatment with cardioselective beta-blockers).

			After suxamethonium			
	K_1	K_2	1 minute	2 minutes	4 minutes	6 minutes
Group 1						
A	4.3	4.2	4.4	4.4	4.6	4.7
B	3.8	3.8	3.9	3.9	3.9	3.8
C	4.0	3.9	4.2	4.2	4.1	4.1
D	4.1	3.9	4.0	4.0	4.0	4.5
E	3.9	3.9	4.3	4.4	4.5	4.7
F	3.9	4.0	4.0	4.0	4.1	4.0
Group 2						
G	3.9	3.8	3.9	3.9	4.0	4.0
H	4.2	4.1	4.3	4.2	4.2	4.2
I	4.2	4.1	4.4	4.3	4.3	4.4
J	3.6	3.4	3.5	3.8	3.7	3.8
K	3.5	3.4	3.5	3.6	3.7	3.6
L	4.7	4.4	4.6	4.7	4.5	4.6
Group 3						
M	3.9	3.8	4.0	4.2	4.3	4.2
N	4.1	4.1	4.1	4.1	4.2	4.2
O	3.8	3.8	3.8	3.7	3.9	4.0
P	3.8	3.8	3.9	3.9	3.9	3.9
Q	3.4	3.3	3.2	3.1	3.3	3.4
R	4.2	4.0	4.5	4.6	4.7	4.5

K_1 = level before induction with thiopentone; K_2 = level before injection of suxamethonium.

Table 2. Mean (SD) plasma potassium changes in group 1 (no treatment with beta-blockers), group 2 (treatment with propranolol) and group 3 (treatment with cardioselective beta-blockers).

	Change in plasma potassium mmol/litre				
		After suxamethonium			
	After thiopentone	1 minute	2 minutes	4 minutes	6 minutes
Group 1	−0.05 (0.10)	+0.18 (0.15)	+0.20 (0.18)	+0.25 (0.21)	+0.35 (0.33)
Group 2	−0.15 (0.08)	+0.17 (0.08)	+0.22 (0.12)	+0.20 (0.09)	+0.23 (0.10)
Group 3	−0.06 (0.08)	+0.12 (0.21)	+0.13 (0.31)	+0.25 (0.28)	+0.23 (0.18)

After thiopentone = change from pre-treatment level; after suxamethonium = change from level before injection of suxamethonium.

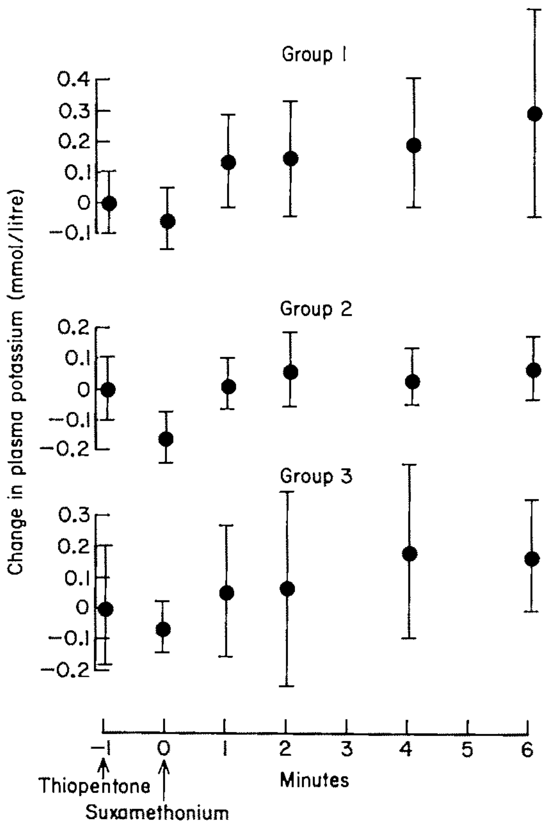

Fig. 1. Mean (SD) plasma potassium changes from pre-induction levels in group 1 (no treatment with beta-blockers), group 2 (treatment with propranolol) and group 3 (treatment with cardioselective beta-blockers).

Table 3. Mean (SD) $Paco_2$ levels in group 1 (no treatment with beta-blockers), group 2 (treatment with propranolol) and group 3 (treatment with cardioselective beta-blockers).

	$Paco_2$, mmHg					
			After suxamethonium			
	Pre-induction	After thiopentone	1 minute	2 minutes	4 minutes	6 minutes
Group 1	42.8 (3.4)	46.3 (3.2)	53.0 (5.4)	49.9 (6.3)	46.0 (7.7)	42.6 (7.3)
Group 2	44.0 (3.7)	48.3 (5.5)	49.4 (6.1)	53.4 (9.1)	46.0 (5.7)	40.5 (6.4)
Group 3	43.8 (7.4)	47.6 (4.8)	51.4 (2.9)	50.1 (6.3)	44.4 (3.7)	41.8 (4.9)

Discussion

The pattern of plasma potassium change seen in this study, of an initial fall during induction with thiopentone followed by a rise after the administration of suxamethonium, is similar to that described by previous workers.[11] The maximal rises of 0.2–0.4 mmol/litre recorded are also in agreement with the maximal figure of 0.5 mmol/litre quoted by Paton.[10]

Nonspecific beta-blockade by propranolol might influence the suxamethonium-induced rise in plasma potassium by interfering with the adrenergic control of sodium and potassium flux across the cell membrane. This could lead to either an increased net efflux of potassium from depolarising muscles, or decreased uptake of the released potassium at other sites. The fact that there were no significant differences in the magnitude of the initial rises seen in the three groups indicates that there is no significant increase in net potassium efflux. This would be consistent with recently published findings that the exaggerated rise in plasma potassium during exercise in beta-blocked subjects cannot be attributed to greater release by active muscle.[12] It is possible that beta-blockade may still modify the response by prolonging the rise because of impaired clearance of the released potassium from the extracellular space, but a longer period of observation would be necessary to evaluate this point. It should also be noted that the effects of beta-blockade on the much larger rises in plasma potassium which have been recorded following the administration of suxamethonium to patients with burns or denervation injuries[13] remain to be investigated.

Acknowledgment

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Unexplained hepatitis following halothane

Your leading article (*Anaesthesia* 1986; **41:** 575–8) was more restrained than some writings on the subject, but it did not have the courage to emphasise that it is *still* an *unexplained* pathology. A greater disservice to good, scientific anaesthesia was done in the winter 1985 Journal of the Medical Defence Union. It stated *Halothane hepatotoxicity exists. Claims for damages on this fact are increasing.*[1] Such remarks encourage unjustified litigation and tend to make young anaesthetists afraid to use what has been shown, in many millions of cases, to be a simple and safe drug. They will feel constrained to use a different technique, which may well be more hazardous, but, apparently, less liable to litigation. One example suffices.

Frail old ladies of 80 years and more can be anaesthetised very successfully for hip operations with a small amount of halothane and a lot of oxygen, while they breathe spontaneously (I often wonder whether the relative hepatic hypoxia, from all the nitrous oxide which is often mixed unnecessarily with halothane, produced some of the unexplained hepatitis we are discussing). But I have known a young anaesthetist, in order to avoid UHFH in such a patient, paralyse her, intubate her and ventilate her with 8 litres/minute, or more, of dry nitrous oxide and oxygen. He may have avoided the risk of being blamed for giving the lady halothane hepatitis, but he ran the risk of giving her a sore throat, a dried trachea, broken teeth, inhaled teeth, death from disconnexion in a paralysed patient, trouble with a complicated scavenging system and awareness during the operation, not to mention the problems of positive pressure ventilation with a closed chest in a paralysed patient.

The figure given for deaths following UHFH is 1 : 10 000, which is exactly the same as the figure given for the risk of death solely as the result of anaesthesia,[2] and Stock and Strunin[3] sum up their recent review article with the words 'At present there is no test that is specific for halothane-induced liver damage. Therefore the diagnosis of UHFH may only be made by excluding all other causes of postoperative liver dysfunction'. This is not a very scientific way of influencing a choice of anaesthetic, and Strunin and Davies[3] write that 'on emotional, rather than scientific grounds, most authorities feel that a second halothane anaesthetic should not be given 'within several months'. What these authorities seem to ignore is that, *before the introduction of halothane*, Brunson *et al.*[4] wrote about the increasing prevalence of unexplained liver necrosis. How would these authorities explain unexplained liver necrosis today, had halothane not arrived? And do they remember that one of the earliest reports about the possibility that halothane might cause hepatitis appeared, not in a medical journal, but in an American business publication,[5] at about the time that Abbott Laboratories introduced methoxyflurane as a competitor?

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Alternative medicine

At a time when 'alternative medicine' is very much in the news, it is a coincidence that Dr E.N.S. Fry (*Anaesthesia* 1986; **41:** 661–2) and ourselves should both be studying the anti-emetic action of acupuncture or acupressure.

On a visit to the People's Republic of China one of us was impressed by the use of a modified form of acupuncture as a prophylaxis against vomiting during early pregnancy. It was challenging to see whether this could be studied in as scientific a method as other clinical trials. We have used it with three opioid premedicants (each with a different profile of onset and duration of action) in a standard situation in which patients are scheduled for minor gynaecological operations and receive a standard anaesthetic. Some of our findings have been reported[1–5] and these are summarised in the accompanying Figure. It was not possible to do a double-blind evaluation, but patients with each of the premedicants shown received, at random, either the drug alone or the drug followed by manual acupuncture at the P6 (Neiguan) point. In the case of nalbuphine premedication a dummy acupuncture point was also used. Neiguan is just above the right wrist between the tendons of palmaris longus and flexor carpi radialis and a 1.2-cm 30-SWG stainless steel needle was used and manipulated manually

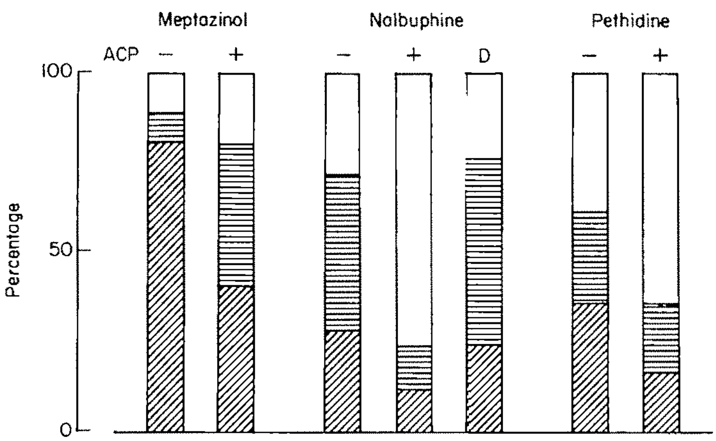

Fig. 1. Histogram of combined results. ACP, Acupuncture: +, applied; –, not applied; D, dummy acupuncture. ▨, Vomiting; ▤, nausea.

for 5 minutes. The occurrence of a non-anatomically distributed feeling of heaviness, numbness or tightness verified the correct position of the needle. The dummy point, at the right elbow, was chosen because it does not lie on any recognised acupuncture lines.

We had 25 patients in each of the series and with each opioid we were able to demonstrate a statistically significant reduction in peri-operative emetic effects (from 90 minutes before to 6 hours after operation) but no similar effect with the dummy point acupuncture. When the results are added together the difference is very striking; the incidence of vomiting was halved and the number of patients with neither nausea nor vomiting was doubled by the use of acupuncture.

Contrary to Dr Fry's findings, this method of acupuncture does seem to produce some degree of anti-emetic action for at least 8 hours after administration. Whether this can be prolonged by using electrical stimulation for 10 or 15 minutes we do not know, nor do we know whether similar results can be obtained by acupressure alone.

We have had no side effects from this in our experience, which now extends to well over 250 therapeutic acupunctures. Our next attempt is to see whether acupuncture can be used therapeutically in the management of sickness from cancer therapy.

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Alcuronium, pancuronium and phaeochromocytoma

In our recent article on phaeochromocytoma, the anaesthetic management was based on the use of drugs that do not stimulate the sympathetic system.[1] Despite reports of hypertensive responses after pancuronium,[2] we used it because it had been considered the agent of choice in this condition.[3] This is a report of severe hypertension and tachycardia after its administration in a patient with phaeochromocytoma whom we have recently anaesthetised.

A 44-year-old man was scheduled for resection of a phaeochromocytoma after pre-operative preparation with oral phenoxybenzamine and propranolol. Premedication was with fentanyl 0.30 mg and scopolamine 0.5 mg intramuscularly, one hour before operation. A radial artery was cannulated under local anaesthesia. Induction was performed with thiopentone 300 mg and fentanyl 0.45 mg, followed by pancuronium 8 mg and lignocaine 100 mg intravenously. The patient's lungs were ventilated with a facemask for one minute, and then the arterial blood pressure suddenly increased from 110/60 to 240/135 mmHg and the heart rate from 65 to 105 beats/minute. There were no noxious stimuli and no catecholamine-releasing drug, apart from pancuronium, was administered; laryngoscopy was not started. A sodium nitroprusside infusion restored the blood pressure to 140/90 mmHg, and propranolol up to 1 mg was necessary to control the heart rate to 76 beats/minute.

Approximately 4 minutes after haemodynamic stability was regained, additional fentanyl 0.40 mg and lignocaine 100 mg were given. Tracheal intubation was accomplished successfully and no significant blood pressure or heart rate changes occurred. Several hypertensive crises were controlled with nitroprusside and phentolamine during tumour manipulations, and fluid overloading and dopamine were necessary to restore the blood pressure after tumour removal.

It has been suggested that the type of muscle relaxant used in phaeochromocytoma is of secondary importance.[4] Nevertheless, this report, and similar ones, do not support this opinion. Pancuronium may stimulate catecholamine release from the sympathetic nerve terminals.[5] In addition, it may also inhibit catecholamine re-uptake by the postganglionic nerve endings, leading to an augmentation of the sympathetic response.[6] Its administration produces a moderate increase in blood pressure and heart rate,[7] and these effects may be exaggerated in the presence of a phaeochromocytoma.[2] The muscle fasciculations that suxamethonium causes may lead to catecholamine release from the tumour, and therefore its use is not recommended. Gallamine is contraindicated because of its sympathetic effects, and tubocurarine is known to stimulate histamine release which can cause catecholamine release. The new non-depolarising relaxants atracurium and vecuronium may be attractive alternatives. However, atracurium may provoke histamine release[8] and vecuronium may cause a small increase in catecholamine levels,[9] and both facts need consideration. On the other hand, alcuronium has ganglionic blocking properties, and produces moderate hypotension by a reduction of the vascular resistance.[10] Furthermore, it has been demonstrated that tracheal intubation with alcuronium was associated with lack of catecholamine increase and pressor response.[11] However, for unknown reasons, the use of alcuronium in phaeochromocytoma has been undervalued.

There are two different clinical situations during the resection of a phaeochromocytoma; the first is related to the increased catecholamine concentration in response to noxious stimuli or surgical manipulations in the presence of the tumour, and the second is connected to the decrease of catecholamine that follows tumour removal. From a theoretical point of view the concept of an ideal muscle relaxant is illusory, since the best agent for one situation may not be so in another.[8] Phaeochromocytoma is a clear illustration of this statement. Therefore, in future we propose to use alcuronium until the tumour resection, followed by small doses of pancuronium, when necessary, once the tumour has been removed.

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Blood tests before elective surgery

It is routine in many hospitals to do blood tests, such as haemoglobin, haematocrit and serum creatinine, before operation for all patients scheduled for elective surgery. We studied 4506 patients who were scheduled for elective surgery in our hospital. The natural hypothesis is that many of these otherwise healthy

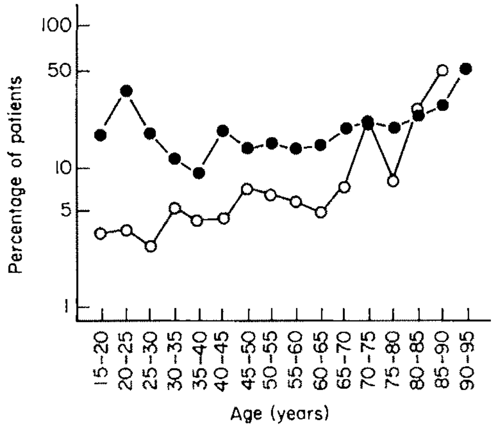

Fig. 1. The percentages of ill (●) and healthy patients (○) with haemoglobin less than the lower limit of the reference range (11 g/dl) by age (years).

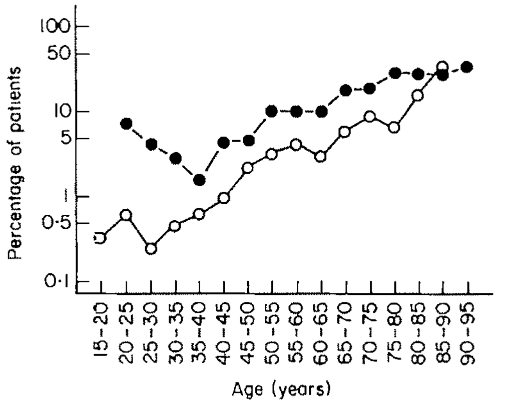

Fig. 2. The percentages of ill (●) and healthy patients (○) with serum creatinine above the upper limit of the reference range (120 mmol/litre) by age (years).

patients should have blood values within the normal range. It might therefore be possible by their ommission both to avoid the inconvenience to the patients and to save the budget for these blood tests without endangering the patient.

The patients were divided into two groups according to simple criteria: patients were classified as ill on the basis of daily need for drugs (apart from minor tranquillisers or the contraceptive pill), insulin or oral hypoglycaemic drugs, or the presence of a diagnosis of neoplastic disease. Of the patient sample, 1099 were defined as ill and 3407 as healthy. The deviations of values for haemoglobin, packed cell volume and serum creatinine from the reference ranges employed by this hospital were calculated (Figs. 1 and 2). It was concluded that determination of these variables is unnecessary in healthy patients under the age of 50 years. However, the information desired about these values must, of course, depend upon the extent and nature of the planned surgery. In addition, one parameter of haemoglobin or packed cell volume is sufficient, since it is possible to calculate the other as haematocrit = 0.07 + 0.04 × haemoglobin mmol/litre (haemoglobin g/dl × 0.6206 = haemoglobin mmol/litre) in the reference range.

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Epidural buprenorphine for postoperative analgesia

It was very interesting to read the article by Wolff *et al.* (*Anaesthesia* 1986; **41**: 76–9) about the use of epidural buprenorphine for postoperative analgesia after major orthopaedic surgery. They concluded that 0.3 mg buprenorphine administered epidurally provided similar analgesic properties when compared to 4 mg morphine, and did not offer any advantage over morphine for postoperative pain relief. Their results also show that unwanted side effects were more frequent in the morphine group.

A similar study of 100 patients in which 0.3 mg buprenorphine was given epidurally after orthopaedic surgery on the lower limbs for postoperative pain relief, has been done here. My results are comparable to those of Wolff *et al.* No major unwanted side effects were seen in the series.

Wolff *et al.* do not mention any problems, even theoretical ones, that may be caused by epidural buprenorphine. There were more side effects after morphine and the authors suggest that buprenorphine does not offer any advantage over morphine although there is the theoretical possibility of respiratory depression which may be difficult to reverse with an opiate antagonist. Unless or until this is reported, buprenorphine appears to have definite advantages over morphine as their study suggests.

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A reply

We thank Dr C.M. Kumar for his stimulating interest in this subject. Our experience with this partial opiate antagonist is quite extensive,[1–4] both in terms of time and the number of patients we have treated. This drug has become our alternative epidural narcotic analgesic when treating cancer pain.

Buprenorphine, as mentioned, differs from morphine pharmacologically with a modified receptor relationship, a high degree of lipid solubility and a long elimination half-life.[5] Given as Temgesic® 0.3 mg/ml, diluted as indicated, we know that the solution contains no potentially neurotoxic preservatives or stabilisers. Differences in side effects are usually ascribed to receptor kinetics. However, recent studies have revealed, when compared with equipotent dosages of morphine chloride,[6] a similar degree of respiratory depression, which makes respiratory monitoring essential for at least 12 hours following the first epidural bolus. Any respiratory depression is readily reversed with an injection of doxapram (1 mg/kg).

Owing to μ-receptor antagonism, symptoms of opioid abstinence may arise when this analgesic is changed to a pure agonist. We agree with Dr Kumar that, in the present study, the small number of unwanted side effects suggests the use of buprenorphine as the first choice for epidural pain treatment.

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Another modification of the Nuffield 200 ventilator

We read with interest the article by Dr M.N. Calhaem (*Anaesthesia* 1986; **41:** 631–4). We would like to present a simpler system which has been in use at Bridgend for the past three years. The figure shows the system. Note that the Newton valve of the Nuffield 200 ventilator is now separated from the main body. It is connected to the metal part of a Penlon Bain breathing system which is modified. The disposable coaxial tubing is lengthened by 50 cm to 2 metres, in order to prevent contamination of the fresh gas flow during controlled ventilation. The exhaust port of the Newton valve is in direct connexion with the bag-mount and adjustable pressure-limiting (APL) valve, the fresh gas inlet remaining discrete. The system requires only one specially machined part and has proved to be both reliable and convenient in use.

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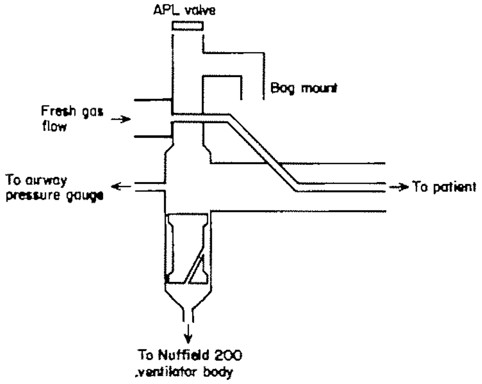

Fig. 1. Modified Nuffield 200 ventilator.

A reply

Thank you for the opportunity to reply to this letter. The system described by Dr Kwan and Dr See is similar to mine in using the patient valve of the Nuffield 200 ventilator to isolate the reservoir bag and adjustable pressure limiting valve during inflation of the patient's lungs. Moving the patient valve to the Bain system inlet block appears to cause several problems. Firstly, as Dr Kwan and Dr See have stated, it is necessary to lengthen the Bain coaxial tubing to prevent dilution of the anaesthetic gases. Henville and Adams[1] have implied that 500 ml (the volume of 1 metre of standard corrugated tubing) is necessary to prevent mixing. The volume of the outer lumen of the standard Bain coaxial tubing is 300 ml/metre and so this could indicate the need for an additional length of 1.7 metres, i.e. 3.2 metres in all.

Secondly, the attachment of the patient valve appears to be to the port that would normally accommodate the reservoir bag, and the bag and adjustable pressure limiting valve are on a new structure. It is not clear if the machined part is incorporated, but presumably it is, in order to accommodate these changes. It is, in my experience, the machined parts that add considerably to the cost.

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Reference

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A hazard of silencers for jet ventilators

This is a caution about the use of low resistance moisture and heat exchange filters as 'silencers' for high frequency jet ventilators as suggested by Dr Conacher (*Anaesthesia* 1986; **41**: 882) and reports a case where the use of such a device in a manner similar to that advocated almost led to the death of a patient.

A 68-year-old man was admitted to the intensive therapy unit after he had fallen from a ladder. He sustained multiple rib fractures as his major injuries. He was initially managed conservatively with a thoracic epidural for pain relief and controlled oxygen therapy. His condition, however, deteriorated over the next 48 hours as his underlying pulmonary contusion worsened, and he developed respiratory failure, which required ventilatory support of his lungs. In view of the dangers of precipitating a pneumothorax by use of conventional intermittent positive pressure ventilation, it was decided to use high frequency jet ventilation with its associated advantages of lower peak airway pressures. The patient's trachea was intubated with a Hi-Lo jet ventilator tracheal tube (Mallinckrodt) and his lungs were ventilated with a high frequency jet ventilator (Acutronic). Normal blood gases were maintained with a driving pressure of 1.7 bar, an inspiratory–expiratory ratio of 40% and a respiratory rate of 150 breaths/minute: the resultant minute volume was 18 litres. Humidification of inspired jet gases was achieved using the Acutronic humidification system and water delivery adjusted according to the manufacturer's nomogram.

The machine was exceedingly noisy and at the suggestion of the registrar a low resistance moisture and heat exchange filter was placed at the expiratory limb of the T-piece as a silencer, a common practice in theatre use. The patient's condition worsened after 3 hours. He became hypotensive, with a tachycardia and there was a deterioration in blood gases. A pneumothorax was suspected, and was confirmed by chest X ray. Insertion of an intercostal drain led to a resolution of the pneumothorax, but the patient remained markedly hypotensive, despite significant colloid replacement. The filter was removed from the end of the T-piece and there was an immediate improvement in blood pressure and haemodynamic variables. The filter was found to be saturated with water. It was not possible to measure the resistance of the filter at the time but it was impossible to blow through it.

We surmise that the pneumothorax and hypotension were secondary to excessive intrathoracic pressure, up to 1.7 bar, as a result of outflow obstruction to the ventilator system caused by the saturated filter. This filter was placed at the expiratory limb of the T-piece and not at the connector of the tracheal tube as suggested by Dr Conacher, but this latter position may be associated with an even greater risk of pneumothorax and hypotension, since obstruction of the filter subjects the patient to the full pressure of the driving gas without allowing dissipation of pressure within the T-piece entrainment system.

As a result of this incident, I consider that such devices should not be used as silencers for jet ventilators; however, if they are then frequent inspection and change of filter is advocated in order to avoid the problems encountered in this case.

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Selective bronchial intubation

Your readers may be interested in an extension of the above technique (*Anaesthesia* 1986; **41:** 519–26) which the writer has found useful in children. The well-known technique of the use of the Fogarty balloon is employed but the end of the balloon catheter, which has a malleable stilette, can be bent to resemble the shape of a standard bronchial suction catheter. Correct placement after the balloon has been inflated can be checked in the usual way by auscultation.

This has been used successfully in two children; one of 6 years with an empyema and the other of 10 years of age with a lung abscess. On both occasions, the biliary catheter was passed through the rubber bung of the Magill suction union blindly into the main bronchus. Soiling of the dependent lung was prevented in both cases.

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Difficult bronchial intubation

The recent correspondence about bronchial intubation prompts us to describe our technique for dealing with this problem. If difficulty is encountered performing blind bronchial intubation, an adult Negus bronchoscope is introduced into the appropriate main bronchus under direct vision. Oxygenation is maintained with the Sander's injector and anaesthesia ensured with incremental doses of induction agent at suitable intervals. A long lubricated bougie is inserted into the chosen bronchus. The bronchoscope is then removed and an appropriate single lumen bronchial tube is railroaded down the bougie into position. This is facilitated by an assistant supporting the proximal end of the guide, and if a fairly large diameter bougie is used the tube will pass into the lung without kinking in the oropharynx.

There are a number of techniques currently advocated for difficult bronchial intubation. A rigid intubating bronchoscope may be used but the field of vision with this instrument is in practice very limited and easily obscured by secretions. The use of an adult Negus bronchoscope overcomes this problem and even an inexperienced bronchoscopist will usually intubate the correct bronchus without difficulty. The fibre optic bronchoscope is used by some clinicians and although the field of vision is excellent, manipulation of the instrument into the required position needs considerable practice. In addition, oxygenation is difficult to maintain during the procedure and smooth passage of the tube is often impeded when the flexible bronchoscope kinks in the oropharynx.

Our technique is most often used after failure to position a double lumen tube blindly. However, it is particularly useful when accurate isolation of a single lung is critical, as in the presence of a bronchopleural fistula. This method is simple, safe and uses equipment readily available in all thoracic operating theatres. We strongly recommend it as an alternative to the standard techniques described.

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Diamorphine in aqueous solution

The comments made by Dr Reynolds on the subject of diamorphine in sodium chloride solutions (*Anaesthesia* 1986; **41:** 554–5) were interesting. In general terms I agree that similar results can be obtained with continuous intravenous infusions of morphine as with diamorphine, but this may not be the case in all applications of the latter. There is, for instance, evidence that the disposition of diamorphine administered epidurally is more favourable than that of morphine.[1] The extremely high water solubility of diamorphine hydrochloride also commends its use in preference to the stable salts of morphine when large doses are required to be administered in small volume injections, or by portable continuous infusion devices.[2]

There are two particular points in Dr Reynolds' letter with which I take issue. Firstly, it is stated that since *it is well known that diamorphine hydrochloride in aqueous solution degrades spontaneously at room temperature* . . ., then clinicians administering diamorphine by slow continuous infusion have simply struck on a novel way to administer morphine, since morphine is the product of this hydrolysis. In support of this statement Martindale[3] is cited. Scrutiny of the references quoted in Martindale[4–7] does indeed confirm that diamorphine is hydrolysed in aqueous solutions at room temperature, but the time course involved, whilst relevant to pharmacists, is inconsequential to the clinician. The time in which 10% of the diamorphine originally present in aqueous solutions is degraded is variously quoted as 31 days at 20°C,[3] 10 days at 25°C,[2] and 4–6 weeks at 22°C.[4,5] None of these papers reports on the stability of diamorphine in 0.9% sodium chloride; however, a variety of buffered and unbuffered aqueous solutions were investigated, and in no case was significant degradation found to occur in a period as short as 24 hours.

A more recent study[8] investigated the disposition of diamorphine administered to patients by various routes. A high performance liquid chromatographic assay capable of determining accurately between diamorphine and its metabolites to a concentration of 12.5 ng/ml was employed. No degradation of diamorphine dissolved in saline was detected on samples from an infusate before or after a 180-minute infusion period.

Secondly, the statement made in Dr Reynolds' letter that diamorphine is not a more potent form of morphine, but merely a more quickly acting form, is open to debate. Most diamorphine will indeed be rapidly converted *in vivo* to 6-monoacetylmorphine and morphine, but the differences in potencies and lipid solubilities of these metabolites are likely to influence their disposition in favour of neural tissue in the crucial distribution phase. This will occur whether or not the drug is administered by infusion, and the result is that more morphine-like activity is seen in the central nervous system after an equimolar dose of diamorphine, than after morphine.[9] It must, however, be accepted that except in strictly molar terms, morphine and diamorphine have equal efficacy in most applications.[10]

Until new work is published on the stability of diamorphine in 0.9% sodium chloride, at relevant clinical concentrations, using a sensitive and specific assay, firm conclusions cannot be reached. However, at present the balance of probability suggests that a clinician can expect virtually complete stability from a solution of diamorphine over a period of, say, 24 hours. Appropriate uses of diamorphine by infusion should not be abandoned because of unsubstantiated fears of its degradation in sodium chloride or other aqueous solutions.

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Modified Magill forceps for difficult tracheal intubation

We would like to suggest a modification to the Magill forceps which facilitates nasotracheal intubation. The modification enables a firmer grip on the tracheal tube and a new way of transmission of the force compared to the original. The distal part of the forceps now consists of a fork and a simple lever with which it is possible to grip the distal end of a nasotracheal tube firmly in the oropharynx and to manipulate it with sure and precise movements through the vocal cords into the trachea. The most important advantages of the new forceps are that with little movement of the hand the tube can be moved some distance and the tube can also be bent as and when necessary.

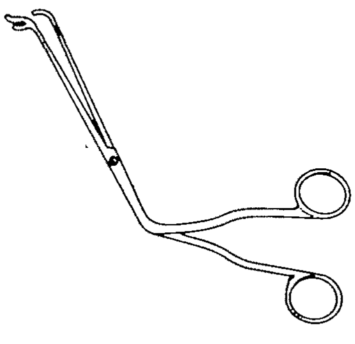

Fig. 1. Modified Magill forceps.

We find the modified forceps extremely useful in cases in which the tube fails to advance easily and particularly when the tip of the tube strikes the upper trachea, as happens quite frequently, due to the anatomical position of the glottis. The new forceps can be made in different dimensions (patent pending).

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Simple device for the assessment of local anaesthetic blocks in children

Local anaesthetic nerve blocks may be used for postoperative pain relief in both adults and children. The assessment of the duration and quality of the analgesia may be made in adults by questioning the patients and by the use of the linear analogue pain scale, but this is more difficult with young children and impossible with infants.

In a recent study, a simple measuring apparatus was devised and used in an attempt to quantify the success and duration of two different local anaesthetic techniques given to children and infants for postoperative pain relief. The apparatus allowed quantifiable data to be obtained and so may be of value to others undertaking similar studies. The device comprises an empty 50-ml intravenous infusion bag connected by a one-metre length of manometer tubing to an aneroid gauge reading pressure in mmHg. The infusion bag contains 30 ml air; this is sufficient to prevent the side walls from touching when the bag is compressed, but insufficient to cause a positive reading at rest.

The application of the device involves placing the bag over the incision site, which is analgesic from a local anaesthetic block, and applying pressure to the wound through the bag. When any discomfort is indicated by the patient the reading is noted in mmHg from the aneroid gauge.

The apparatus was successfully used in a study of the analgesia provided by local anaesthetic blocks for surgical incisions in the inguinal region. The technique required initial questioning and assessment to ensure that the patient was not already experiencing pain from the incisional site, after which the bag was gently compressed against the skin over the incision until the child complained of any discomfort. Patient participation was encouraged by interposing the child's hand between the bag and that of the observer as the pressure was applied. It was possible with such participation to detect when the child was beginning to feel discomfort by any reluctance to press harder and to ensure that excess pressure was not applied unnecessarily.

The advantages of the pressure bag technique over a pinprick estimation of the success or failure of a local anaesthetic block are that it is non invasive, it tests the effectiveness of the block to prevent wound pain rather than the skin analgesia that is tested in a pinprick technique, it enables a quantitative prediction of the duration of the block if used serially, and the bag moulds itself to the contour of the skin and allows readings to be taken in the presence of thin surgical dressings.

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Difficulties with tracheal suction

A wide variety of specialist tracheal tubes are now available for use in intensive care. We recently experienced a problem of apparent incompatibility between a tracheal tube and suction catheters.

A 35-year-old man, with a severe head injury and deteriorating respiratory function, had his oral tracheal tube exchanged for an 8-mm thermosensitive PVC Hi Lo nasotracheal tube (Mallinckrodt) after 48 hours. This was accomplished without difficulty, but when suction was attempted the catheter (Portex 12FG, angled tip) would not pass down the tube even when water and KY jelly were used as lubricants. A chest X ray confirmed correct tube placement. Further attempts were made over a period of 2 hours to pass a suction catheter, but despite trying two other catheters (Portex 14FG, straight; Mallinckrodt Gentle Flo 14FG, angled tip) none would go through. Finally, the tube was removed and replaced with a Portex nasal tube through the same nostril. Suction was then accomplished easily.

Experimentation with this tube, and with another unused one from the same batch, revealed that the inner surface was sticky and it was only with the greatest difficulty that suction catheters could be passed. Passing a suction catheter was made more difficult by acute angulation of the tracheal tube, such as would happen when used for nasal intubation, and by increasing the temperature of the tube to body temperature. If a catheter was passed successfully it was sometimes only with the greatest difficulty that it could be removed. Increased pulling caused increased sticking.

Surface adhesion of polymers such as polyvinylchloride (PVC) has been the subject of detailed investigation.[1] Plasticisers are used by both Portex and Mallinckrodt to soften the PVC, which in its native state is hard and inflexible, but they also increase its tendency to surface distortion. Shear forces at the surface cause the polymer to deform, and dramatically increase friction. Adhesive forces also exist as a result of electrostatic bonding between the polymer surfaces. These characteristics make clingfilm work but they are clearly unsuitable properties in a tracheal tube meant for use in intensive care. The PVC used in Portex tubes contains a silicone oil, dimethyl siloxate, which leaches onto the surface and acts as a lubricant to prevent this problem.

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Limitations of the Grass FT03 transducer

In their report on anaesthesia for Friedreich's ataxia (*Anaesthesia* 1986; **41:** 296–301), Bell *et al.* monitored the effects of muscle relaxants using a Grass FT03C force displacement transducer. Freund and Merati[1] have noted that this transducer is intended for small animal studies and will give erroneous data if used in humans, since its capacity will be greatly exceeded. In particular, a train-of-four response may be interpreted as showing no fade with the FT03 transducer when fade would be detected with an adequate transducer. Furthermore, the time to apparent recovery of first twitch may be seriously underestimated since the maximum output from the transducer will occur even when full twitch height has not been reached.

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Reference

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A reply

Thank you for the opportunity to reply to the letter by Dr Sosis. We are aware of the paper by Dr Freund and Dr Merati. It is, however, our experience that using an FT03 force displacement transducer does not pose a problem using the train-of-four technique in the human subject. It should be recognised that the work carried out by Dr Freund and Dr Merati on the FT03 and FT10 force displacement transducers involved the use of tetanic stimulation which generates a greater force than the train-of-four.

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Haemonetic cell saver

We were interested to read of Dr Kumar's use of the Haemonetic cell saver as a method of autotransfusion in a Jehovah's witness undergoing aortobifemoral grafting (*Anaesthesia* 1986; **41:** 774–5). Our experience of 110 cases, of which 23 were for aortic surgery, leads us to make several comments.

Dr Kumar is to be congratulated on being able to return 950 ml of washed red cells after salvaging only 1200 ml of blood from the wound. Assuming a starting haematocrit of 45%, this is perfect conservation, something we have never managed to achieve. We have an average return of 60% of lost red cells during elective aortic surgery, or about 500 ml of washed red cells (haematocrit >55%) for every litre of salvaged blood of 45% haematocrit. With smaller total losses and well directed surgical suction, a higher return may be possible, as Dr Kumar suggests.

We prefer to follow the recommendations of the manufacturers by using a double suction tube so that saline heparinised with 30 000 units/litre is added to the blood as it enters the tip of the sucker. There can be no clotting in the tubing at low aspiration rates, but the volume in the reservoir will be slightly increased by whatever saline has been added.

Our experience of surgery involving massive blood loss has been that coagulation problems have not been as common, since the number of units of bank blood transfused has been reduced. A protocol which combines a smaller number of units of bank blood with cell-saver autotransfusion, supplemented by appropriate component therapy with fresh frozen plasma and platelet concentrate, has now been adopted. Despite the large dose of heparin used in the suction part of the cell saver, the washing of the cells is so effective that a systemic heparin effect cannot be detected by thrombin time estimation in the patient after use of the machine.

The comment about the risk of particulate and air emboli may apply to some methods of autotransfusion but is not a criticism of the cell saver, which can now be set up to allow reinfusion by techniques similar to routine infusion of bank blood bags.[2]

Despite the obvious appeal of this machine in the management of patients refusing transfusion of bank blood, it cannot be applied with equal success to all forms of surgery. It is only really effective when aspirating blood that has pooled, for example, during arterial surgery, and does not allow effective salvage from a generalised ooze such as found in a mastectomy wound. Nor ought it to be used in surgery of malignant tumours for fear of disseminating malignant cells.

Furthermore, by the time that sufficient blood has been obtained for processing, a significant loss of blood will have occurred and, in patients likely to suffer large losses, this is usually replaced initially by bank blood. Where this is not possible, use of plasma substitutes will lead to early haemodilution. Later, the reinfused red cells (with a packed cell volume of 55–60%) cause a rise in haemoglobin concentration.

These reinfused cells are suspended in saline so that during massive blood loss, even if red cell salvage were complete, there would be a progressive loss of plasma protein, causing a potentially hazardous reduction in oncotic pressure and in clotting factors.

We have reported elsewhere[3] our experience of the cell saver system of autotransfusion, which has proved of significant help in the management of cases with massive blood loss, with a marked reduction in the consumption of bank blood. There have been no complications attributable to the use of the machine.[4] Its successful use requires well organised cooperation between the technician (in our hospital, a senior ODA) working the machine, the surgeon using the suction and the anaesthetist assessing the loss and supervising its replacement.

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The unit of applied force

The use of certain SI units can obscure important findings. Dr Kumar and Dr Messahel (*Anaesthesia* 1986; **41**: 880) state that an epidural catheter may break if a tension greater than 20 N is applied. But how many anaesthetists can translate this into something comprehensible?

A more serious example concerns the application of cricoid pressure. In the Editorial on the Confidential Enquiry into Maternal Deaths we are reminded[1] that considerable force must be applied to the cricoid to prevent regurgitation of gastric contents. Dr Wraight and colleagues[2] have demonstrated that a force of at least 44 N is necessary. This paper is mentioned in many of my Part I FFA lectures, and although many trainees have read it, I have not yet found anyone who could comprehend the force. However, once the force is converted to the familiar 4.4 kg wt (and a pair of kitchen scales is provided to press upon) this valuable paper ceases to be meaningless.

Please may the kg wt be included also as a unit of force in publications?

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Is your scavenging working?

Our hospital is equipped with BOC Mark 1 active scavenging unit which operates at a nominal flowrate of 25 litres/minute. For 6 years following installation, this equipment received no routine servicing. However, since 1984 Ohmeda (BOC) have decided to institute onsite servicing involving stripping and cleaning, at a cost of £31.00 per annum, per unit.

We decided to check the flowrate in these units by occluding the bottom of the collecting reservoir with a plastic bag and elastic band and placing a Wright's respirometer at the inlet (where the anaesthetic system is connected). Of the 12 locations that we checked, we found that only three indicated a flowrate greater than 20 litres/minute and six showed flowrates below 10 litres/minute (see Table). These results imply that in six locations, the scavenging system would not effectively dispose of anaesthetic gases, especially if a ventilator were in use. We showed that this could be rectified by replacing the flowmeter on the scavenging system, and readings above 20 litres/minute were now obtained (see Table).

We conclude that the servicing of individual units in the field does not maintain their specification. A better approach is to make the simple measurement described above and arrange for service exchange of the flowmeter whenever the indicated flowrate drops below an acceptable value. It is to be hoped that Ohmeda will support this maintenance policy by the provision of service exchange units.

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R. RAILTON
J. MILTON
R.A. DAVIE

Location	Existing serviced unit flowrate (litres/minute)	New unit flowrate (litres/minute)
A	6.8	22.5
B	7.1	22.5
C	6.3	23.9
D	4.8	23.4
E	8.9	24.1
F	5.4	22.5

A contaminated Marcain ampoule

We would like to draw your readers' attention to an ampoule of 0.5% bupivacaine, which was discovered by one of our midwives preparing to top-up an epidural for pain relief in labour. This ampoule, sterilised and packaged by the manufacturers, had a visible white precipitate at the bottom, but no other obvious defect. However, close inspection under a microscope revealed a hair-line crack which had enabled evaporation to occur and sodium chloride to crystallise out (see Figure).

This appears to be an isolated case, but it reinforces the need for close checking of local anaesthetic ampoules for epidural or intrathecal use, to ensure that contamination has not occurred.

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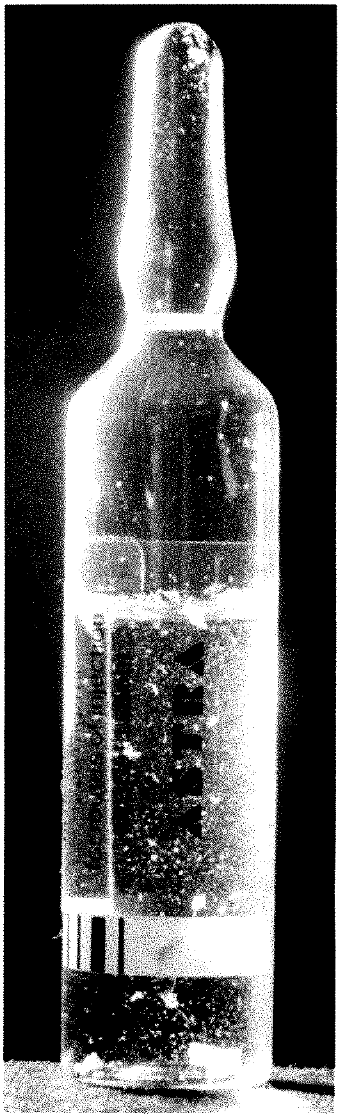
J.C. Sugden
J.L. Bembridge



A reply

We welcome the opportunity to comment on this letter. All manufacturers of ampoules take the greatest care to eliminate any cracked or leaking ampoules from a production run before final packing occurs but clearly subsequent trauma can result in cracks appearing. It is difficult to imagine how a crack of this size could have been missed by the inspection process. The point is that all ampoules should be inspected before use but we do not know the cause of this defect.

The presence of a sterile blister around the ampoule prevents contamination of the contents and the introduction of Steripacks® for Marcain ampoules must be a significant improvement in product protection. This ampoule could have become contaminated by micro-organisms without this packaging.

The precipitate in the ampoule was of bupivacaine hydrochloride salted out as a result of the increased sodium chloride concentration, and not sodium chloride.

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A.K. Watson

Minitracheostomy

'When breathing is difficult and time is short', skilled minitracheostomy may indeed overcome otherwise insurmountable upper airway obstruction and 'save a life' (quotes from the current advertisements). However, it concerns me that those of us with less than the requisite skills and experience, should contemplate insertion of a 4.0 mm internal diameter cannula in this manner, as the method of first choice in such a desperate situation. My own limited experience with the technique suggests that rapid success is not guaranteed in the best of circumstances.

If ventilation is possible by no other means, and options are available, perhaps one should pause for a moment to consider whether needle cricothyrotomy[1] is not a wiser alternative. The presence of difficult regional anatomy, and imperfect posture, lighting or assistance, are but a few other factors that may influence this critical decision on how best to establish emergency transtracheal ventilation.

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Reference

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Multipurpose breathing systems

The modification of the Bain system (*Anaesthesia* 1986; **41:** 631–4) which allows a switch between spontaneous and controlled ventilation would appear to be a useful simplification of a Mapleson D system. However, Dr Calhaem appears to suggest that such an arrangement is more advantageous than other multipurpose systems, and refers to the ADE system. This inference should be viewed in the light that the essential function of Calhaem's modification offers no improvement over that of the ordinary Bain system for spontaneous ventilation and that all the disadvantages of the latter still obtain. The ADE system avoids all these problems since it offers the combined advantages of a Mapleson A system for spontaneous ventilation with those of the Bain for controlled ventilation.

It has been demonstrated clearly that, to prevent rebreathing during spontaneous ventilation, Mapleson D systems require up to three times the fresh gas flow compared to Mapleson A systems.[1,2] This results in substantially higher gas and vapour costs (especially if isoflurane is used) and reduced humidification of inspired gases.[3]

Dr Calhaem avoids reference to the improved version of the ADE system[4] which is almost as simple to use as his modification. A major feature of the current ADE system is that detachment of the ventilator or reservoir bag and closure of the expiratory adjustable pressure limiting valve are not now necessary; only those components required during spontaneous or controlled ventilation are included in the system while all unnecessary parts are *automatically* excluded but remain attached to the system. Thus, the anaesthetist merely selects the A or E mode of use by a single lever *without further adjustment.*

In addition, there is now substantial evidence that the ADE system is more efficient for spontaneous ventilation than the Magill,[5,6] such that an average fresh gas flow of 3–4 litres/minute is all that is required. This compares to flows of 8–12 litres/minute with the Bain.[6] Furthermore, the ADE system can be used not only with ventilators that power a Bain system, but also with minute-volume divider ventilators such as the popular Manley series.[7] Other advantages have been reported independently.[8] While Dr Calhaem's suggestion has its application, it is not functionally as economical or practical an alternative as the ADE system.

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A reply

Thank you for the chance to reply to this letter. In terms of gas and vapour consumption, a Mapleson A is a better system than a Bain for spontaneous breathing. However, a considerable number of anaesthetists choose a Bain system for spontaneous breathing for practical reasons: it is lighter, narrower and more flexible and, given a higher gas flow, functions quite adequately.

Having accepted the use of a Bain system for both spontaneous and controlled ventilation, my apparatus was designed to ease the changeover between the two and to add several safety features. To change modes with a normal Bain system requires removal of the reservoir bag and replacement with ventilator supply tubing and closure of the expiratory valve. Dr Humphrey's ADE system does not require components to be moved but does require the operation of a lever-controlled mechanism. This has two disadvantages. Firstly, it is possible for the lever to be in the wrong position, with poor consequences for the patient. Secondly, the mechanism, needing to be airtight, requires machining to close tolerances. This must add considerably to the cost of the Humphrey ADE system, whose current price is £251.90 excluding VAT. My device has no levers or machined parts and could, I believe, be constructed for tens rather than hundreds of pounds. In addition, it has the added safety of leaving the bag in the system during controlled ventilation and the pressure gauge in the system during spontaneous ventilation.

To cling to the doctrine that a Mapleson A system *must* be used for spontaneous breathing during anaesthesia requires a system such as Dr Humphrey's where a lever must be incorporated, with the disadvantages outlined above. It is perhaps more acceptable to suffer the increased gas consumption and gain the benefit of lower capital cost, increased safety with no lever and the practical advantages of a lightweight system.

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Recovery times

It is easy to accept Dr Binning's assertion (*Anaesthesia* 1986; **41:** 786) that recovery times with methohexitone are comparable to those achieved with propofol, because of his considerable experience of termination of pregnancy. My continued advocacy of propofol is based on morbidity studies[1-3] conducted with some 600 patients attending for termination of pregnancy, and our results are comparable with those of other workers in terms of patients' assessments of their postoperative morbidity. However, the replies provided by patients when leaving hospital and returned by patients on the day following the termination are, for patients receiving propofol, entirely different from those provided by patients who had other intravenous induction agents. Patients' comments that they were hungry, that they should have been fed sooner and that they were able to return to normal activities earlier, represent an entirely different quality of recovery for this procedure. Our experience with methohexitone is that patients complain of headache, nausea and drowsiness on the same night, and are seen by the ward staff to be unhappy when they awaken. It is this difference which, in my view, commends propofol for this procedure.

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Bradycardia and hypotension in spinal anaesthesia

The letters from R.J. Purnell (*Anaesthesia* 1985; **40:** 1245) and C. McLoughlin (*Anaesthesia* 1986; **41:** 554) about bradycardia during spinal anaesthesia seem to have overlooked basic physiological principles. Even though paralysis of cardiac sympathetic fibres plays some part in the production of bradycardia, the effect is very variable and there is no correlation between the height of the block and degree of bradycardia; there should be, if bradycardia were due mainly to block of cardiac accelerator fibres.

There is, however, a direct relation between changes in arterial pressure and pulse rate which is unrelated to the height of analgesia. Arterial blood pressure is maintained during spinal anaesthesia mainly by cardiac output which, in turn, is lowered if venous return decreases. When this happens the heart responds to the reduced atrial filling by slowing. This reflex, which is mediated through intrinsic chronotropic stretch receptors in the right atrium and ventricle, is dominant during spinal anaesthesia over the carotid sinus and aortic arch baroreceptor reflexes which produce the compensatory tachycardia when blood pressure decreases.[1] Drug therapy and premedication influence pulse rate, but any pronounced bradycardia under spinal anaesthesia *must* be taken as a sign of a decrease in venous return, and a decrease in arterial blood pressure.

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Anaesthesia, 1987, Volume 42, pages 90—94

Book reviews

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Clinics in Anaesthesiology. Preparation for Anaesthesia

Edited by J. Stevens. Pp. 798. W.B. Saunders Company, 1986. £13.50.

A cursory glance at this book reveals an impressive list of contents, suggesting that we might expect a storehouse of goodies, many of which are scattered throughout the 350 pages of text; unfortunately, too many are stale. Claims about careful revision should set the tone of expectation, yet somehow the name of one contributor is misspelt and another is entirely omitted. There seems to be a lack of liaison between the publisher, the editor and the contributors as to the nature of their brief, and rival series have little to fear from this competitor.

It is difficult to assess the potential readership for this volume and it must therefore be assumed that it is directed at examination candidates. The sections on the paediatric patient and unusual paediatric conditions are well presented, but that on unusual medical conditions contains inaccuracies and omissions. It is sadly dated and it therefore comes as no surprise to be told that 'ether should be avoided in hyperthyroidism'. There are other irritating descriptions of disease which are unhelpful, such as that on hydatid and Chagas' disease. Methohexitone is missing from the list of drugs to be avoided in epilepsy.

The Editor has deleted the contributions he made on monitoring and the safety of anaesthetic equipment which originally appeared in the first edition. Three experts on local anaesthesia are now huddled together in one chapter to make way for an admirable section on the assessment of the patient with cardiovascular disease. Other newcomers consider the patient with renal disease, a chapter without references, while that on liver disease can only muster 30% of its references from the 1980s. Respiratory disease is another disappointment, since the chapter is almost a complete replica of that which appeared in the first edition in 1980. There are comprehensive lists of drug interactions and, yet again, I considered that I could still be reading the first edition. Chunks of text have been transferred from one edition to the other without correction. To describe a 1978 reference as being recent in the 1980 edition of this book is fair, but to repeat the same comment in 1986 is inept.

The pre-operative management of diabetes is inadequate, albeit one is advised to consult Alberti, whose regimen is monotonously trotted out at examinations. Hall's critical assessment should have appeared in print, as well as a consideration of the other hazards that face the diabetic patient being prepared for surgery. I suppose there must be a reason why the new insulins only appear in the chapter entitled 'Anaesthesia during pregnancy', a section that contains some updated material to distinguish it from its predecessor, but omits the problems of tocolytic therapy and the hazards of intravenous non electrolye solutions during labour.

One regularly subscribes to *Clinics in Anaesthesiology*. Some volumes are like vintage cars and are to be treasured, while others lack sparkle and polish. My main concern is to find someone suitable to whom I can safely donate this review copy.

L. Kaufman

Clinics in Anaesthesiology. Obstetric Analgesia and Anaesthesia I and II

Edited by G.W. Ostheimer. Pp. 232 and 444. W.B. Saunders Company, 1986. £13.50, £13.50.

There are now many books on obstetric anaesthesia, written by authors from both sides of the Atlantic. These two volumes are different in that, under the editorship of Dr Ostheimer, American, British and Australian experts deal with the many problems associated with anaesthesia and pregnancy. Overall, however, there is a bias towards the American viewpoint in that 80% of the contributors come from either the United States or Canada. This does not detract from their value for British readers since, during the past decade, there has been a growing uniformity of opinion on how to deal with problems associated with obstetric anaesthesia.

Volume I deals with the principles of obstetric anaesthetic care and the physiological and pharmacological basis is covered in a detailed manner. The chapters on the pain of parturition, perinatal pharmacology of anaesthetic agents and the toxicity of local anaesthetic drugs are excellent and give the reader an up-to-date knowledge of these topics. It was disappointing to find that the British work on the use of H_2-receptor blockers was not referred to in depth in the chapter on gastric aspiration. Paracervical block is rarely used in British obstetrics because of the risk, to the fetus, from amide-type local anaesthetics, and the drug recommended here, chloroprocaine, is not available in Britain. The British contributions on general anaesthesia, failed intubation and spinal anaesthesia are of high quality and the hazards of each are clearly defined. The warning in the section on intraspinal narcotics that such treatment should still be regarded as experimental is timely and emphasises the danger of delayed respiratory depression (a serious error in the dose of sufentanil appears in Table 1 on page 150).

The second volume is devoted to the clinical management of various common and rare conditions encountered in obstetric anaesthesia. The choice of essential anaesthesia in early pregnancy is discussed and reference is made to Nunn's work on the possible danger of nitrous oxide. In the chapter on heart disease, the value of a slowly induced lumbar extradural block is emphasised and the use of invasive monitoring described. Disorders associated with pregnancy, some more common than others, such as hypertension, obesity, diabetes and neurological disease are considered and recommendations for their anaesthetic management given. Very practical chapters on the treatment of haemorrhagic states, amniotic fluid embolism and breech and multiple pregnancy are included and these are of great value to all engaged in obstetric practice. This volume concludes with an international overview of obstetric anaesthesia. The great difference in maternal mortality between developing countries and the United Kingdom is underlined. Means of improving this situation are discussed and the author stresses the need for education and self-reliance as well as financial aid.

The two books make a very valuable addition to obstetric anaesthetic literature. They should be read by both anaesthetists and obstetricians, who will find much of value to enhance their patient care. The extensive and up-to-date references provided by every contributor will make them essential for those engaged in research and teaching. Both are well produced, illustrated and excellently edited and represent good value for money.

J.A. Moore

Essential Anaesthesia

By F.R. Ellis and I.T. Campbell. Pp. 130. Blackwell Scientific Publications, 1986. £6.95.

The extent of undergraduate teaching in anaesthesia varies considerably between different universities in the United Kingdom. Perusal of the published curricula reveals that clinical attachments to anaesthesia vary from 0–4 weeks with an average of approximately 2 weeks. Clearly, however, the extent of teaching considerably exceeds these officially allocated levels, since anaesthetists are frequently invited to contribute lectures during the undergraduate course in physiology and pharmacology, to take ward rounds during surgical attachments and to provide additional tuition in cardiopulmonary resuscitation. There is no doubt that these latter activities are amongst the most valuable contributions towards the undergraduate curriculum.

During the clinical attachment to anaesthesia, what should undergraduates be taught? Many believe that students should not be instructed on how to administer anaesthetics, but that the greatest emphasis should be laid upon pre-operative assessment and preparation for surgery, postoperative management of pain and fluid and electrolyte balance, the acquisition of practical skills such as maintenance of the airway and insertion of intravenous cannulae and, of most importance, theoretical and practical instruction in cardiopulmonary resuscitation.

With these objectives, and with the marked variation in exposure to anaesthetic teaching, what should a prospective author incorporate in a textbook suitable for medical students in British universities? One would suggest a brief description of all the activities in which anaesthetists are involved so as to indicate clearly to undergraduates the scope of anaesthetic practice. This is imporant if we aim to compete in recruiting the best students into our specialty. In addition, I would lay greatest emphasis on the topics described above and provide sufficient detail as to offer an incentive for the purchase and retention of the text.

How does *Essential Anaesthesia* meet your reviewer's concept of an ideal undergraduate textbook? It conforms well in many respects. The full scope of anaesthetic practice is described well in a most readable manner, albeit briefly in a text of 120 pages, devoid of illustrations. In such a small book, it has not been possible to expand at length upon topics essential to the preregistration houseman. For example, premedication occupies a little more than one page, no practical details of local anaesthetic blocks or cannulae insertions are provided, postoperative pain occupies just over one page, fluid and electrolyte balance and blood transfusion occupy only three pages, etc.

Whether or not a student decides to purchase a copy will probably depend on the length of his clinical attachment in anaesthesia. However, those choosing to buy one of the larger undergraduate texts available should easily and profitably read *Essential Anaesthesia* on the first day of their clinical attachment to anaesthesia. In addition, this book should be placed on the recommended reading list for Schools of Nursing.

G. SMITH

Parenteral and Enteral Nutrition

By G.D. PHILLIPS and C.L. ODGERS. Pp. 272. Churchill Livingstone, 1986. £13.95.

The authors' intentions are to introduce the subject to an interested but uninformed reader, so that he may be able to decide upon the indications for, and anticipate some of the complications of, enteral and parenteral nutrition. It is said to be the third edition of the book, although the previous two editions were printed for private circulation.

It provides a very clear summary of the biochemical consequences of the nutritional deficiencies to be met with in sick patients. It goes on to describe in short chapters each of the factors to be considered; carbohydrate, protein, fat, vitamin and mineral requirements are described separately. The requirements of the fetus, neonate and child are discussed, as is also nutritional support in malignant disease and pregnancy. A short section is devoted to electrolyte and acid-base balance. Two of the longer chapters relate to pharmaceutical aspects of parenteral and enteral nutrition and there are other sections describing access problems and the types of catheters used for both enteral and parenteral routes. Throughout the book it is emphasised that the enteral route is to be preferred if it is usable. The book finishes with appendices containing lists of the suppliers of such equipment and tables of relevant biochemical data.

The book is very well put together, clearly printed and very easy to read. Because many of the chapters are very short and the index is comprehensive, the answers to most of the questions asked by anyone unfamiliar with these nutritional problems can easily be found. Those who require more detailed information would probably not buy a book of this type but each chapter ends with a helpful list of recommendations for further reading. There are many books on the market which have the same aims as this one but, in the writer's opinion, this is one of the best.

J.C. STODDART

Anesthesiology 1986

Edited by T.H. STANLEY and W.C. PETTY. Pp. 286. Martinus Nijhoff, 1986. £33.25.

Anesthesiology 1986 promised to be an exciting addition to the plethora of reviews, annuals and digests in the specialty. The authors are recognised experts, such as Covino on local analgesia and spinals, Strunin on the liver and hepatitis, Shapiro on brain protection and cerebrovascular surgery and Grundy on evoked potentials and the management of spinal cord function. In the event, the book is disappointing. The material is taken from the refresher course manuscripts of the 31st Annual Postgraduate Course in Anesthesiology at Utah in February 1986, but the selection of the subjects for publication appears to be arbitrary and disjointed. The editing is very poor. For example, papers are included which refer to tables and figures not to be found in the text. Massive differences in style are permitted, so that even a chapter (on brain protection) which comprises a series of lists and figures without any text, is permitted.

The chapters vary in quality and reflect current thinking in the United States of America. This is particularly evident in the approaches to the use of neuromuscular blocking agents, and also outpatient and paediatric anaesthesia. Near misses and risk management are subjects of great importance, but the general recommendation of eternal vigilance and good training may not be enough. They do echo the advice of the British Medical Defence Union. Most common preventable incidents in the quoted Boston study involved the breathing system, inadvertent drug administration and drug overdose. This has not been the experience of several studies elsewhere.

The book is a hardback but is, surprisingly, presented with a typewriter-like print which is difficult to read. Despite the high quality of a few chapters, *Anesthesiology 1986* cannot be recommended as a high priority for purchase.

R. GREENBAUM

Anesthesia

Edited by R.D. MILLER. Pp. 2419. Churchill Livingstone, New York, 1986. £135.

This second edition of Miller's monumental textbook has been expanded to three volumes and now includes detailed and wide-ranging topics in the basic biological and physical sciences relevant to anaesthesia and intensive care.

Volume 1 is divided into four sections. Section 1 consists of an account of the history of anaesthesia and a detailed chapter on the legal aspects of anaesthetic practice. The American position is very evident in this section and the British reader will find that there are still, fortunately, some advantageous differences in the legal attitudes between the two countries.

The second section, entitled 'Scientific foundations of anesthesia', contains, in addition to a chapter on general pharmacology (which for some reason is separated from the other chapters on the biological sciences), readable chapters on physics (including breathing systems), computers and statistics.

The third section covers the assessment and preparation of the patient for anaesthesia and surgery. Throughout this section there is a commendable emphasis on safety and the reduction of anaesthetic risk. Many aspects are covered in quite extraordinary depth; for example, the mathematical analysis of cost–benefit data on pre-operative investigations. Whether or not this sort of thing excites you, the degree of detail (including, in the 103-page chapter on intercurrent disease, more than 700 references) is impressive.

The final section has chapters on monitoring, intubation, and several on the detailed general pharmacology of inhalational agents and intravenous narcotics. The comprehensive nature of this volume makes it difficult to find a topic which is not adequately covered, and often from a refreshingly new angle.

Volume 2 includes close examination of the physiology and pharmacology of the neuromuscular junction, the autonomic nervous system, and the cardiovascular, respiratory, hepatic and renal systems, as well as chapters on acid-base balance, fluid therapy and cerebral blood flow. Clinical anaesthesia is represented by chapters on regional anaesthesia and anaesthesia for thoracic, cardiac, vascular and neurosurgery and neuroradiology. More than twenty authors make contributions to this volume and it is both to their credit, and to the Editor's, that the style is consistently both scholarly and readable. There is, inevitably, an emphasis on American practice; not all drugs mentioned are available in the United Kingdom and some, such as Althesin, have been withdrawn since the book was written.

The subject of anaesthesia related to specific systems which commenced in Volume 2 is completed in *Volume 3*. This group of sections has a wide range of subject matter, both in quantity and diversity. It ranges from anaesthesia for patients with liver disease to the function of the recovery room. The depth to which each topic is discussed is variable; there is a whole section on anaesthesia for carotid endarterectomy, which could have been included under anaesthesia for vascular or neurosurgical procedures. The size of this one section is in fact longer than the whole subject of anaesthesia for trauma surgery! One finds it hard to believe that the length of these sections reflects the importance of these two forms of surgery. Whilst being fully satisfied with most aspects of the coverage of these topics, one is somewhat surprised that the subject of difficult intubation in ear, nose and throat surgery occupies merely half a page.

The remainder of the volume is divided between specific anaesthetic problems and techniques, consultant anaesthetic practice (which relates primarily to treatment of chronic pain and cardiopulmonary resuscitation) and a final section on critical care medicine. There has been a marked enlargement of this last section since the first edition and it now represents a comprehensive view of all aspects of intensive care in the United States. The chapter on thoracic anaesthesia is particularly good and contains more practical information on pre-operative evaluation and the placement of bronchial tubes than some specialised textbooks.

Dr Miller is to be congratulated on combining together so many authors for this large work without excessive repetition. Each chapter is liberally supported by relevant references up to 1984, as well as a number of older more classical references. An indication of the size of this particular task can be seen from the fact that the index alone is some 66 pages long! Allowing for some transatlantic differences in anaesthetic practice, these volumes thus maintain their reputation as a most valuable and informative route of entry to the literature. The three volumes make up a masterpiece and will continue as one of the standard textbooks in anaesthesia in the world. There are relatively few fortunate individuals who can afford to purchase all three volumes, but the complete set is essential for departmental and reference libraries.

J.N. HORTON
M. HARMER

The History of Blood Gases, Acids and Bases

By P. ASTRUP and J.W. SEVERINGHAUS. Pp. 332. Munksgaard, 1986. £23.25.

This lavishly illustrated book gave great pleasure to the reviewer. It is a compendium of the discoveries and activities of the long list of great men of physical and clinical science who have contributed to the unfolding and revealing of the blood gas and acid-base story. The book is, itself, a monument to the detective work of the senior author, Paul Astrup, who, aided by John Severinghaus, has provided the reader with a lucid, informative and interesting insight into the minds and lives of these pioneers. Paul Astrup contributed the first seventeen chapters to the book dealing with the period from B.C. up to 1953, and John Severinghaus wrote the last chapter, describing the modern instrumentation developments of the past 30 years.

The early chapters (1–6) are concerned with the discoveries of carbon dioxide, oxygen, and the composition of water, and with the growing interest in the respiratory gases and their measurement in the blood. A whole chapter in this section is devoted to the early blood gas pumps and to the controversy regarding gas secretion. Chapters 8 and 9 are devoted to the oxyhaemoglobin dissociation curve and to carbon dioxide content in the blood. Chapters 10–17 then unfold the history of acids and bases and the development of medical acid-base research and practice right up to the polio epidemic in Copenhagen in 1952–53.

The final chapter, by John Severinghaus, is entitled 'AHA'. This title epitomises 'the millisecond event during which two unconnected thoughts connect—in a new and creative way'. One cannot help but feel sorry for poor Severinghaus. He had gathered together 100 pages of notes including 421 relevant references, and had intended this last section of the book to occupy 200–300 pages, not counting illustrations. Instead, he was gently told to reduce his part of the book to 'about 10 pages' (finally, he achieved 32 pages, including illustrations). There is therefore much left unsaid in this last section of the book, but Severinghaus has now fortunately published this modern history saga in a series of articles in an international journal. Perhaps this editorial decision is justified, as it can be argued that 30 years is but a 'moment' when compared with the previous centuries, but this modern history is still full of interest (with twinges of sadness in places) even if it is not spiced with the intrigue, plotting and scandals of the early forefathers.

Astrup has called his section of the book a 'novel' and it is certainly true that his description of the characters makes them spring from the pages. What he has written is nonfiction, but the history does unfold as a plot. The towering philosphers, physicists, chemists and physicians (to use Severinghaus's phrase) who have contributed to the acid-base and blood gas story are a mixed (and at times motley) bunch. The scores of scientists long revered by a succession of students, in association with famous laws and theories, turn out to be very human after all. Some were grossly arrogant and vain, some were humble and reticent to the point of being recluses, some stole the theories and results of others, some 'doctored' their results, and many were mistrusted and disliked by co-workers and colleagues. Others were plainly good, dedicated honest workers. The interpersonal relationships between many of these scientists and physicians are fascinating to the reader, as are the disputes and character assassinations (right up to the deathbed). All this is brought to the fore in an easy and interesting style.

As Astrup sagely points out, the reader will recognise his colleagues and associates, as well as his 'rivals', within these historical cameos. To quote his phrase, 'a laboratory is a sportsground where the sole object is to be first in a competition—for the sake of honour'. Nothing has changed over the centuries and who knows who will be next to join this roll of honour. Many, if not most, of the pioneers were not accepted in their own time, because their ideas and theories were outside conventional thought, and so there is still hope for the modern outsider or heretic; he or she will already be following a well-trodden and familiar path, if this book is anything to go by.

As for the price, this reviewer was surprised that such a densely packed and lavishly illustrated book could be produced at such a reasonable price. It is highly recommended and each anaesthetic library should have one copy, at the very least.

C.E.W. HAHN

Health Equipment Information: Number 157 (August 1986)—Evaluation of Infusion Pumps and Controllers

The sixth report includes evaluation of the Vickers Treonic C30 Controller, Graseby MS2000, Vial SE200B and Vickers IP4 syringe pumps.

Health Equipment Information: Number 155 (June 1986)—Evaluation of ECG Recorders

Full reports on single channel recorders (Bosch EKG501A and Cardiacare C1201) and three channel recorders (Fukuda Denshi FD36 and Triamed Minicor M3) are now available.

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Central venous pressure measurement

By G.D. Parbrook, P.D. Davis and E.O. Parbrook (*Royal Infirmary, Glasgow*). Tapeslide lecture. Buy or hire from Oxford Educational Resources Ltd, 197 Botley Road, Oxford OX2 0HE. 28 minutes. 22 slides. (1985).

This programme is an addition to the Glasgow series of tapeslide lectures on clinical physics and measurement. The technique of internal jugular vein cannulation is described with diagrams which clearly show the anatomical relationships of the surrounding structures. Calibration and measurement of central venous pressure and the interpretation of readings are described clearly and concisely. In the latter part of the programme the introduction of the pulmonary flotation catheter and the pressure recordings obtained are described and interpreted with the same conciseness and precision which characterise the rest of the programme. As with other tapeslide lectures in this series, the programme is interrupted at intervals to allow the student to self-test his understanding by means of a questionnaire.

The style and presentation are excellent. Although the subject is treated at an elementary level, the clarity is just what is required for an understanding of the anatomy and physiology of central venous pressure measurement by trainees before they receive practical instruction at the bedside.

Minor criticisms can be made. It is a pity that the authors confine themselves to the internal jugular route as the subclavian and arm veins are widely used. The description of pulmonary flotation catheterisation is necessarily limited and might be better expanded in a separate lecture. Nevertheless, the programme will be a valuable addition to the collection of those departments fortunate enough to have a tapeslide library.

J.N. Horton

Nerve blocks around the knee

By K. Brown (*Royal Children's Hospital, Melbourne*). VHS Videocassette. Colour. Buy or hire from BMA/BLAT Film Library, British Medical Association, B.M.A. House, Tavistock Square, London WC1H 9JP. 11 minutes. (1983).

This video describes blocks of the sciatic nerve behind the knee and the saphenous nerve at the medial side of the knee. The good anatomical description lays the foundation for a useful videotape which, coupled with good diagramatic presentation, makes the understanding of the divisions of the sciatic nerve into the popliteal and common peroneal nerve quite clear. The mechanics of blocking them, therefore, become simple and the areas of the leg the procedure is making insensitive to pain are clearly described.

Preference for blocking the common peroneal in the popliteal compartment is emphasised, as when this nerve is blocked at the head of the fibula the incidence of neuralgia is greater. The video then describes the blocking of the saphenous nerve at the knee, emphasising its diffuse nature and warning against the injection of drug solution into the nearby saphenous vein.

The video also indicates the application of these nerve blocks for surgery around the ankle and foot, for pain relief in trauma and for the reduction of ankle fractures. It wisely reminds the operator that as the blocks may last for 12 hours or more, there can be no indication from the patient as to the overtightness of applied plaster casts. This, therefore, has to be guarded against by careful examination of the circulation. This is an easy videotape to follow, but your reviewer considers more attention could have been paid to analysis of the success of these blocks.

K.M. ROGERS

Ankle blocks

By K. BROWN (*Educational Resource Centre, Royal Children's Hospital, Melbourne*). VHS Videocassette. Colour. Buy or hire from BMA/BLAT Film Library, British Medical Association, B.M.A. House, Tavistock Square, London WC1H 9JP. 13 minutes. (1983).

This video describes the anatomy of the five nerves at the ankle that have to be blocked to secure analgesia of the foot and though the anatomical description and illustration of the position of the nerves is good, the video on this subject does not really add much to the information that one can obtain from the current text books.

K.M. ROGERS

Blocks for common paediatric operations

By K. BROWN (*Education Resource Centre, Royal Children's Hospital, Melbourne*). VHS Videocassette. Colour. Buy or hire from BMA/BLAT Film Library, British Medical Association, B.M.A. House, Tavistock Square, London WC1H 9JP. 16 minutes. (1985).

In this presentation the author, Dr Kester Brown, describes the technique of caudal block, penile block and ilio-inguinal block for postoperative pain relief in paediatric surgery. The author starts by reminding us of the anatomy, a sound knowledge of which must be the basis of each of these practical techniques. He does this by means of cadaveric specimens, diagrams, and X rays and apart from the fact that the cadavers are a rather gruesome sight (they are, I am glad to say, not paediatric), coverage of the anatomical details which lie behind these blocks is good.

In describing the caudal block for postoperative analgesia the author covers such necessary information as blood levels of local anaesthetic after the block and the dosage employed. He also states briefly the types of surgery for which the block is useful. In his description of the penile block the anatomical details are particularly well presented. A dosage schedule is also provided and he describes how the block can be done in three different ways, which is commendably comprehensive. His description of the iliohypogastric and ilio-inguinal nerve blocks makes the procedure look easy (which of course it is) and the video presentation shows up very well the importance of using short bevelled needles to elicit the 'pop' on passing through the aponeurosis of the external oblique muscle.

Some people in the UK may criticise the degree of sterility which he employs for the procedure, namely that the operator does not wear gloves but has merely washed his hands and doused them in alcohol solution; the patient's skin is prepared in the same way. Furthermore, perhaps the video could have presented some analysis of the efficiency of these blocks in providing analgesia for the patients postoperatively.

The commentary is clear and the background music uninvasive and although the subjects who have volunteered to be demonstrated on do not appear to be paediatric, the overall result is a video that should be useful not only for demonstrating the procedures to junior anaesthetists, but also for educating nurses and surgeons in the value of these procedures in producing comfort for the patient.

K.M. ROGERS

Lower limb nerve blocks

By K. BROWN (*Educational Resource Centre, Royal Children's Hospital, Melbourne*). VHS Videocassette. Colour. Buy or hire from BMA/BLAT Film Library, British Medical Association, B.M.A. House, Tavistock Square, London WC1H 9JP. 15 minutes. (1983).

The videotape describes the femoral nerve block, sciatic nerve block and the lateral cutaneous nerve of thigh block. It commences with a review of the anatomy both of the origin of the nerves in terms of their roots and also the dermatomal representation on the body surface. The anatomy is described by means of diagrams and by cadaver. The commentary makes reference to the use of either paraesthesiae for identifying the nerves, or a nerve stimulator and it is with this latter piece of apparatus that the sciatic nerve blocks are performed. The importance of the use of a short bevelled needle to identify the passage through fascial planes is mentioned and there is some reference to the drugs employed and their toxic doses.

The anatomical representation of where the femoral nerve lies is particularly good and easy to follow and the description of the technique, employing as it does the distinct feeling of the short bevelled needle passing through first of all the fascia lata and then the fascia iliacus, is very easy to follow. If this method is adhered to, a high success rate will be achieved. The lateral cutaneous nerve block is described in as many minutes as it takes to perform this simple, yet effective procedure.

Three approaches to the sciatic nerve are described. A nerve stimulator is used to indicate that the tip of the needle is near the sciatic nerve. The presentation is clear, both in commentary and vision and is not too long. Good use is made of X rays and scans to describe the compartment within which the nerve lies. The blocks were demonstrated on a young lady subject and her forbearance, if she was not anaesthetised, must be admired.

The reviewer was surprised that the use of adjustable markers on needles, particularly for the sciatic nerve block, was not described; in addition, the advantage of using a nerve stimulator is not made clear. The reviewer would have preferred more discussion concerning the types of drugs used and volumes and dosages employed. This aspect is particularly important as it might be that an observer will decide to execute all three blocks together in the same patient, with the consequent danger of exceeding the toxic limitations of the drugs used.

This is a useful video to introduce students in anaesthesia to the practicality of the regional anaesthetic methods. It would be very useful for demonstrating to nurses and surgeons the advantages of these techniques and how, if theatre lists can be so arranged, they need not use up the limited theatre time available.

K.M. Rogers

Lumbar sympathetic block

By K. Brown and J. Madden (*Royal Children's Hospital, Melbourne*). VHS Videocassette. Colour. Buy or hire from BMA/BLAT Film Library, British Medical Association, B.M.A. House, Tavistock Square, London WC1H 9JP. 26 minutes. (1983).

This video presentation describes the use of lumbar sympathectomy in the treatment of vaso-spastic conditions such as peripheral vascular disease and reflex sympathetic dystrophy.

It starts by describing the anatomy of the sympathetic chain in the lumbar region using a cadaveric specimen for this purpose. It then goes on to describe the procedure and its application in three patients. In the first, a patient suffering from reflex sympathetic dystrophy, it describes how lumbar sympathetic block using local anaesthetic in the early stages of this condition can be curative and when used in the chronic stages of this condition speeds recovery. The video illustrates the diagnosis of the condition, the placement of the needles to deliver local anaesthetic to the sympathetic chain and the measurement of the effectiveness of the block by measurement of the change in skin electrode potential. The second patient illustrates the use of the technique for the treatment of peripheral vascular disease, particularly where rest pain is a feature of that condition. It involves the use of a neurolytic solution of 10% phenol. It includes a mention of the complications that can arise when the technique is performed. The third case is the use of the technique in a child of ten who suffers from reflex sympathetic dystrophy in her leg and here the technique is performed under general anaesthesia.

This is a video that has some value as a general description of the techniques, but would not be complete enough for demonstrating the techniques to somebody who is thinking of employing them himself. For instance, it does not give clearly defined enough landmarks for needle entry points in starting the technique and it does not mention the volume of local anaesthetic used in the cases of sympathetic dystrophy.

The video correctly emphasises the necessity of using the image intensifier along with radio-opaque dye to indicate the position of the needle tip when using neurolytic solution, but fails to mention aspiration of the needle prior to injection. Unfortunately, the X ray pictures are not very clear; they give a poor impression of the placement of the needle tips prior to injection and this is not helpful in what is potentially a dangerous technique.

K.M. Rogers

Burns—nerve blocks of the thigh

By K. Brown and C. Reardon (*Educational Resource Centre, Royal Children's Hospital, Melbourne*). VHS Videocassette. Colour. Buy or hire from BMA/BLAT Film Library, British Medical Association, B.M.A. House, Tavistock Square, London WC1H 9JP. 10 minutes. (1986).

This video is one of a series from lectures on anaesthesia for the burned patient. Its purpose is to describe the use of nerve blocks to provide anaesthesia of the thigh for the taking of skin grafts. It thus describes nerve blocks of the femoral nerve, the lateral cutaneous nerve of the thigh and the posterior cutaneous nerve of the thigh. The descriptions of these blocks are well illustrated by using cadaveric specimens on which the relevant structures have been coloured, diagrams and X rays which employ contrast material to show the distribution of the local anaesthetic around the nerves in their fascial compartment. The blocks are then demonstrated on a patient under ketamine general anaesthesia.

Both at the beginning and end of the tape the author stresses the importance of using short bevelled needles which will elicit the loss of resistance or 'pop' that can be felt when the needle passes through the fascial planes. This is an important guide to positioning the tip of the needle within the fascial compartments that contain the nerves. The principle is well illustrated in the description of the femoral nerve block. Three-dimensional localisation can be achieved with greater certainty by feeling for the two 'pops' as the needle passes first through the fascia lata and then the fascia iliacus, to lie in the compartment in which the femoral nerve runs. The author also indicates that this technique can be used to provide block not only of the femoral nerve, but of the obturator and lateral cutaneous nerve of the thigh as well by instilling a larger volume and persuading the local anaesthetic in a cephalad direction by pressure of the finger distal to the needle.

The video describes three methods of blocking the lateral cutaneous nerve of the thigh, one of which the authors have described as a new approach. The evidence for the adoption of this new technique is provided by a dissected specimen and diagrammatic illustration and will certainly encourage me to try this approach in the future.

The blocking of the posterior cutaneous nerve of the thigh can be accomplished either as part of a sciatic nerve block or on its own as a separate procedure. Again, the positioning of the needle is accomplished by feeling the passage through the superficial fascia and loss of resistance to injection after passing through the muscle layers. Using these three blocks, anaesthesia of the whole of the upper thigh can be achieved, giving excellent conditions for the taking of skin grafts and providing several hours of postoperative pain relief.

This video is a useful one; the only minor criticism is of its presentation. It is a little too quick and because of this one has to stop and rewind to make sure one catches exactly what has been said. The diagram illustrating the new approach to blocking the lateral cutaneous nerve of the thigh requires a couple of rewinds to assimilate its message because the orientation of the diagram is not at first clear. However, these points do not detract from a video that will provide good instruction for junior anaesthetists (and of course surgeons) who wish to acquaint themselves with techniques whose benefit to the patient in providing safe and reliable postoperative analgesia has regrettably suffered from a relative neglect in the schools of anaesthesia in this country.

K.M. ROGERS

Anaesthesia for children

By K. BROWN AND T. SKOROPLAS (*Royal Children's Hospital, Melbourne*). VHS Videocassette. Colour. Buy or hire from BMA/BLAT Film Library, British Medical Association, B.M.A. House, Tavistock Square, London WC1H 9JP. 25 minutes. (1979).

The tape describes in chronological order the handling of infants for surgery from the anaesthetic point of view. It includes, therefore, a description of the preoperative visit, the reception of the child at theatre and the processes required for anaesthesia; it also covers necessary monitoring and finishes with the reception of the child in recovery. It deals with the equipment that is peculiar to paediatric anaesthesia, such as the T-piece circuit, Rendell-Baker masks and the straight-bladed laryngoscope. In a survey of monitoring, it stresses the importance of temperature monitoring and control by means of warming devices, both for gases and the theatre table. It shows the technique of inhalational induction, tracheal intubation, the method of fixing the tracheal tube and the technique of manual ventilation of the lungs. The reaction of neonates to muscle relaxants is mentioned and indeed anaesthesia for neonates is covered separately from the infant.

It is difficult, having viewed this video presentation, to decide for whom it has been made. It is not nearly comprehensive enough, lasting as it does for 25 minutes, to be effective in educating junior anaesthetists. It covers the topics so briefly that they are likely to pick up all the material presented in the video after one or two attendances in the anaesthetic room of a paediatric hospital. In order to educate junior anaesthetists, video presentations would have to be made lasting approximately 10–15 minutes on each of the various aspects that this 25-minute video covers. i.e., premedication, induction, monitoring, recovery and postoperative analgesia. For a complete educational programme on paediatric anaesthesia, video presentations would also have to be made on some of the aspects that this video fails to cover, namely intravenous fluid maintenance and replacement, drug doses and schedules and, more practically, the extubation of the infant, which is an aspect that differs from the adult.

If the video is for nurses who assist the anaesthetist in theatre then it would be useful, although the subject should be introduced by a description of the physiological and anatomical differences between infants and adults.

K.M. ROGERS

Anaesthesia, 1987, Volume 42, pages 99–104

Anaesthetic literature

This section of *Anaesthetic literature* contains references taken from *Current Contents—Life Sciences* for July 1986. The Journals which may be consulted when *Anaesthetic literature* is compiled are those listed in *Current Contents—Life Sciences*, published by the Institute of Scientific Information, Philadelphia, Pennsylvania, USA.

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The collator of this section is Dr L. Kaufman, MD, FFARCS, Anaesthetic Office. The Rayne Institute, University College, University Street, London, WC1E 6JJ. Dr Kaufman is prepared to provide on request, and at a modest charge, a new additional service to our readers. References from January 1984 have been entered on data base. The data are held on Dbase II, and are available on disk, together with a program providing search facilities. Enquires direct to Dr Kaufman at the above address please.

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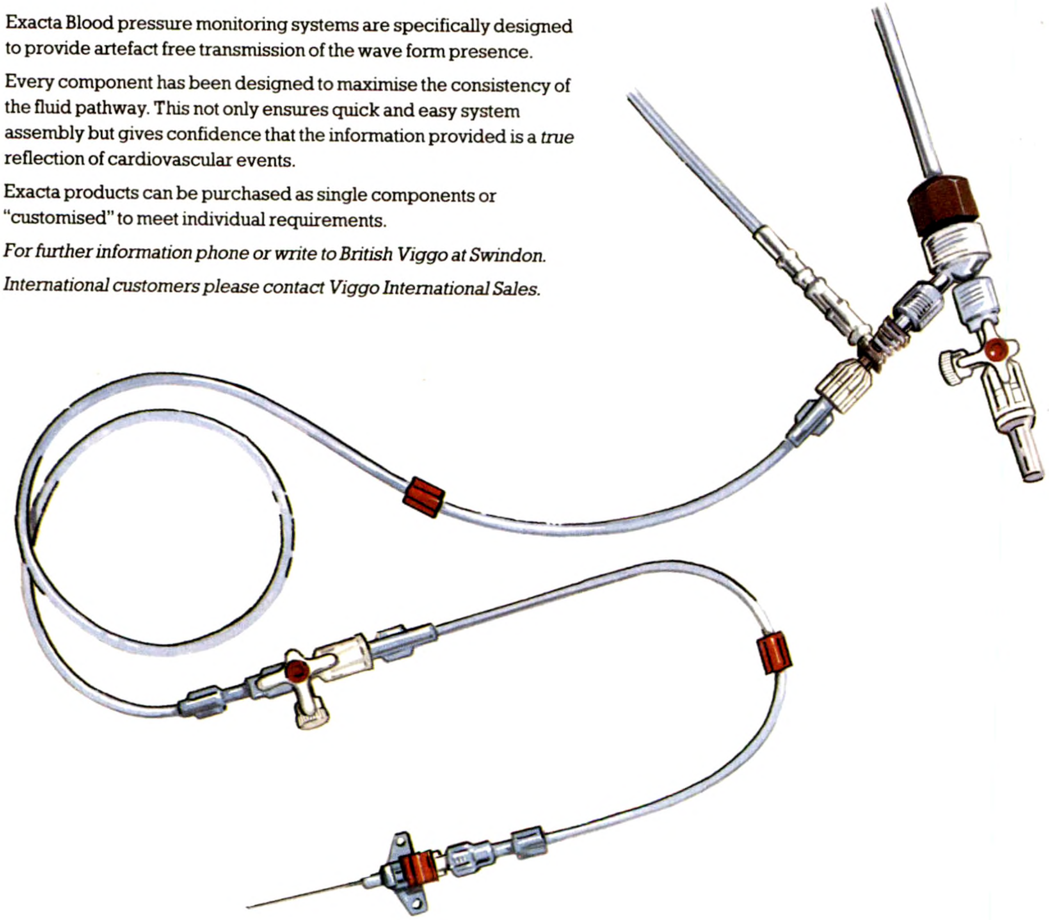

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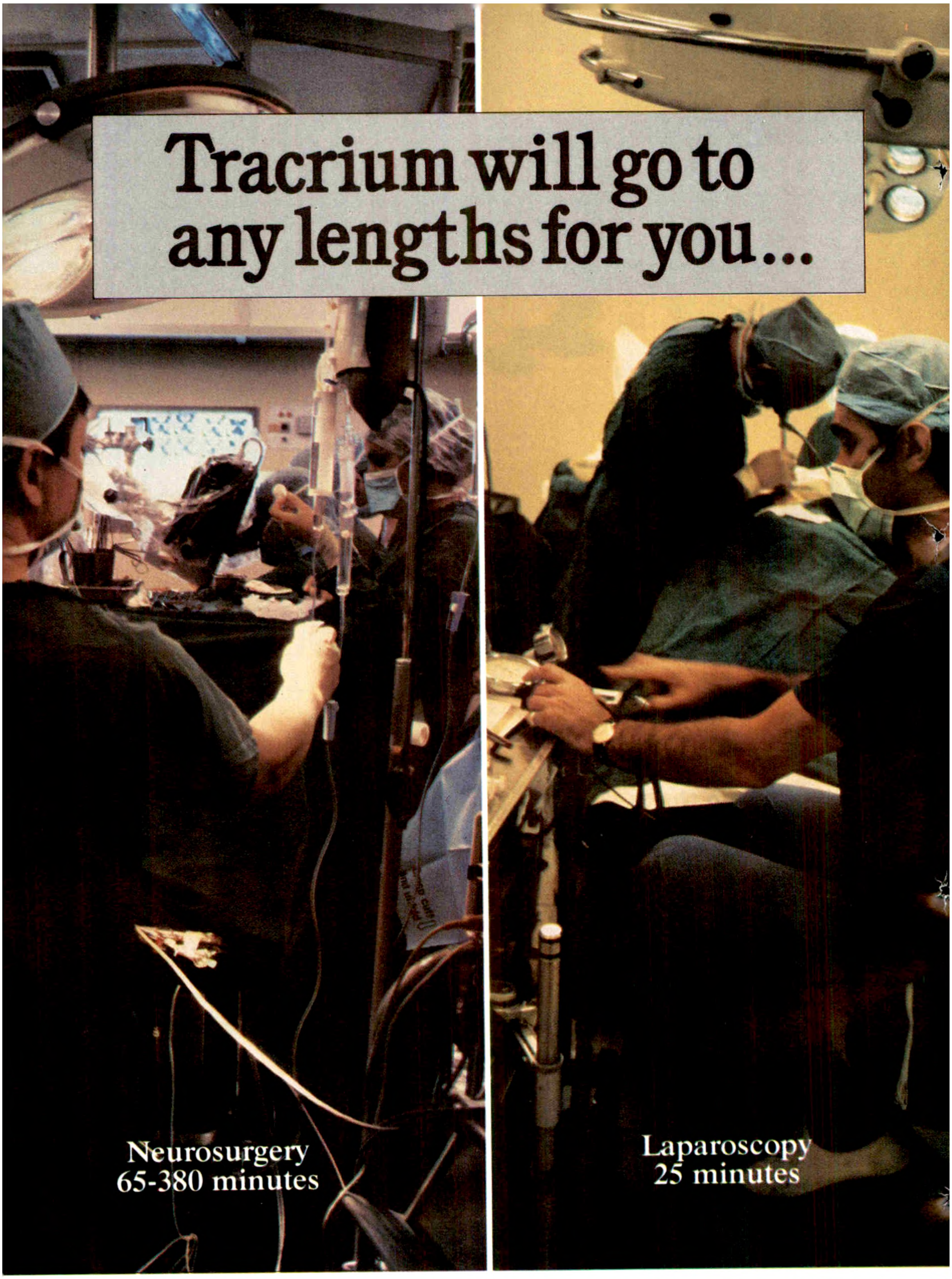

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Dosage and administration

Intravenous injection: Adults and children over 1 month 0.3-0.6 mg/kg depending on the duration of block required; this will provide relaxation for about 15-35 minutes. Tracrium may be used at standard dosage in elderly patients and in those with respiratory, hepatic or renal failure. Full block can be prolonged with supplementary doses of 0.1-0.2 mg/kg as required. Successive supplementary dosing does not give rise to cumulation. Endotracheal intubation can usually be accomplished within 90 seconds of intravenous injection of 0.5-0.6 mg/kg. The neuromuscular block produced by Tracrium can be rapidly and permanently reversed by standard doses of neostigmine which should be preceded by the administration of atropine. Recovery from full block without the use of neostigmine occurs in about 35 minutes.

Continuous infusion: Tracrium is suitable for administration by continuous infusion at rates of 0.005-0.01 mg/kg/minute (0.3-0.6 mg/kg/hour) to maintain neuromuscular block during long surgical procedures. Tracrium maintains acceptable physical and chemical stability in daylight at concentrations of between 0.5 and 0.9 mg/ml at 30°C for up to: 4 hours in: Compound Sodium Lactate Intravenous Infusion B.P.; 8 hours in: Ringer's Injection U.S.P.; Glucose Intravenous Infusion B.P. 5% w/v; Sodium Chloride 0.18% w/v and Glucose Intravenous Infusion B.P. 4% w/v and up to 24 hours in: Sodium Chloride Intravenous Infusion B.P. Tracrium can be administered by infusion during cardiopulmonary bypass surgery, at the recommended infusion rate. Induced hypothermia to a body temperature of 25 to 26°C reduces the rate of inactivation of atracurium, therefore full neuromuscular block may be maintained by approximately half the original infusion rate at these temperatures.

Contra-indications, warnings, etc. Contra-indicated in patients hypersensitive to atracurium besylate. Tracrium should be used with caution in patients receiving aminoglycosides or polypeptide antibiotics and in patients with myasthenia gravis. In common with all other neuromuscular blocking agents, adequate facilities must be available for endotracheal intubation and artificial ventilation. The neuromuscular block produced by Tracrium may be increased by the concomitant use of inhalational anaesthetics such as halothane. Tracrium should not be mixed in the same syringe with any other agent; in particular it must not be mixed with thiopentone or any alkaline agent, as the high pH would cause inactivation of the Tracrium. As with all other neuromuscular blocking agents the possibility of transient hypotension due to histamine release cannot be excluded. Although animal studies have indicated that Tracrium has no adverse effects on foetal development, nevertheless, like all neuromuscular blocking agents, it should be used with caution in pregnant women. Tracrium may be used to maintain neuromuscular relaxation in Caesarean section as atracurium does not cross the placenta in clinically significant amounts.

Presentation Tracrium injection contains 10 mg per ml atracurium besylate. Two ampoule sizes, 2.5 ml and 5 ml are available.

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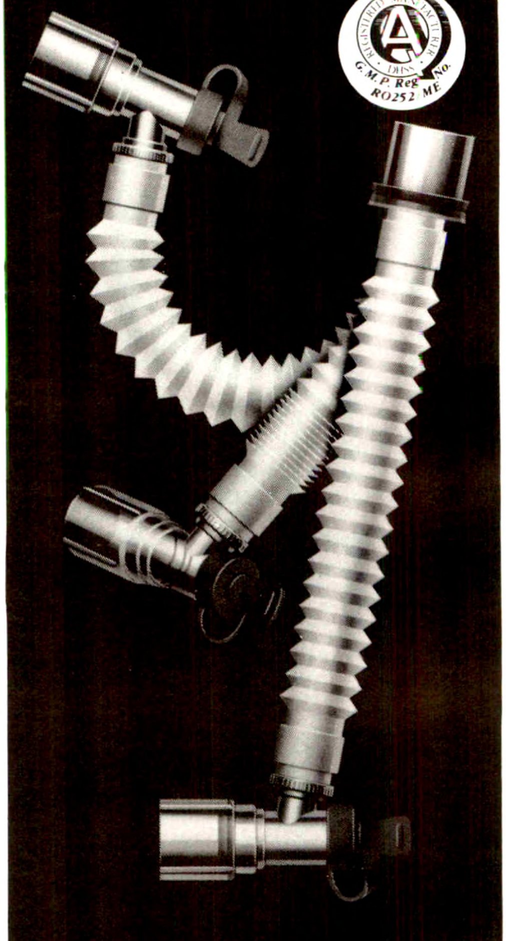

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News and notices

Obituaries

Conway, Cyril Martin, MB, BS, FFARCS, formerly Professor in the Magill Department of Anaesthetics, Westminster Hospital. Qualified from the Charing Cross Hospital Medical School in 1952.

Gilmartin, Thomas James, LRCPI & LM, LRCSI & LM, FFARCSI, FFARCS Eng, DA RCPSI, Hon. FRCSI, late Associate Professor, Royal College of Surgeons in Ireland. Qualified in Dublin 1929. A full appreciation will appear in a later number.

Association of Anaesthetists' Appeal for the Furnishing and Refurbishment of Nine Bedford Square, London

A centre for anaesthesia

Our Appeal achieved its target of £600 000 and closed on 21 September 1986, more than three months ahead of the intended date. Council are extremely grateful for the excellent level of support received from Members. They are also most appreciative of the many donations made by friends in Industry and by Charitable Trusts.

Council wish particularly to acknowledge the contributions of major donors. BOC Health Care have funded both the Museum, which will house the Charles King collection of historical apparatus, and the salary of an Educational Coordinator who will undertake administrative duties for the planned programme of seminars. Janssen Pharmaceuticals, ICI Pharmaceuticals (UK)—who are providing the salary of an Archivist—the Wolfson Foundation, Draeger Medical, Portex, the Wellcome Foundation and Abbott Laboratories have all made major contributions towards refurbishment, furnishing and educational activities. Donors through Analocum Ltd are invited to continue to use this system.

Furnishing is now going ahead due to the success of the Appeal. Members of the Association will increasingly come to appreciate and benefit from the generosity and support of our donors and are more than welcome to visit Number Nine.

Association of Anaesthetists of Great Britain and Ireland

The Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA, England. Telephone: 01–631 1650 (Ansafone service available out of office hours).

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Council 1986–87

All Officers with the exception of the Assistant Editors are *ex-officio* members of Council.

Elected Members: Professor A.P. Adams (*London*), Dr D.S. Arthur (*Glasgow*), Dr W.L.M. Baird (*Glasgow*), Dr Elizabeth Bradshaw (*London*), Dr K. Budd (*Bradford*), Dr J. Edmonds-Seal (*Oxford*), Professor W.S. Nimmo (*Sheffield*), Dr T.W. Ogg (*Cambridge*), Dr J.F. Searle (*Exeter*), Professor G. Smith (*Leicester*), Dr J.A.W. Wildsmith (*Edinburgh*), Dr W.S. Wren (*Dublin*), Dr G.W. Hamlin, Dr Lauren Allan, Dr A.D.J. Nicholl (*Junior Anaesthetists' Group*).

Annual Scientific Meeting, London, 17–19 September 1986

There were 520 registrants at the Meeting which took place at the City University, London. The *Social Programme* was organised by Dr R.H. Ellis (*St Bartholomew's*). A very enjoyable buffet supper was held on board M.S. Naticia which took the participants for a 3½-hour trip up and down the river Thames from Tower Bridge. The annual dinner was held at the Plaisterers' Hall. The toast to the Association was proposed by Viscount Lord Tonypandy to whom the President, Professor M. Rosen, replied and proposed the health of the guests.

Seventy-five firms exhibited in the *Technical Exhibition* and it is again a pleasure to thank them for their enthusiastic participation and to thank Dr R.S. Vaughan (*Cardiff*) who was entirely responsible for the organisation of this important event.

The *Annual General Meeting* was attended by 220 members. The John Snow Silver Medal was awarded to Professor Sir Gordon Robson, CBE (*Royal Postgraduate Medical School*). Honorary membership was also conferred upon Sir Gordon and upon Drs O.P. Dinnick (*Middlesex*), D.D.C. Howat (*St George's*) and C.F. Scurr, CBE, MVO (*Westminster*). Pask Certificates of Merit were presented to Air Commodore A.J. Merrifield (*R.A.F.*), Dr S. Lipton (*Liverpool*), Dr Ruth Mansfield (*Vellore*) and Mr Peter Cull (*St Bartholomew's Hospital*).

This Annual Scientific Meeting was a great success and all credit must go to the organising committee chaired by Dr M.T. Inman (*Plymouth*) as the Immediate Past Honorary Secretary of the Association. The *Scientific Programme was arranged by Professor A.P. Adams* [*Guy's Hospital*) assisted by Dr R.M. Jones. The Association is grateful to them all.

The *Scientific Programme* was as follows.

Session 1: Finance for Anaesthesia and Intensive Care. Chairman: Professor J.P. Payne. Money, anaesthetics and the management board, Mr I. Mills (*Director of Finance Management, Department of Health and Social Security*). Doctors as managers, Mr P. Griffiths (*District General Manager, Lewisham*). Cost benefit analysis in the ICU, Mr J. Tinker (*Middlesex*). Clinical budgeting for anaesthetists, Dr J. Secker-Walker (*University College Hospital*). Anaesthetic equipment: industry's costs, Mr K. Ward (*Ohmeda*).

Session 2: New Concepts in Anaesthesia. Chairman: Professor A.P. Adams. Are the watchdogs sleeping? Chemoreceptors and anaesthesia, Professor R.L. Knill (*Western Ontario*). Bubbles and blood: lessons from NASA and North Sea research, Professor B.A. Hills (*New South Wales*).

Session 3: Acquired Immune Deficiency Syndrome and the Anaesthetist. Chairman: Dr M.L. Heath. Clinical overview and epidemiology of AIDS, Professor M. Adler (*Middlesex Hospital*). Severe lung injury and AIDS, Dr J. Rees (*Guy's Hospital*).

Section 4: New Technology in Anaesthesia and Intensive Care. Chairman: Dr N.I. Newton. The magic of silicon valley: new technology for the anaesthetist, Dr W.R. New (*Stanford, USA*). Multigas electrochemical sensors: state of the art, Dr C.E.W. Hahn (*Oxford*). The anaesthetic machine: what's its future? Professor M.K. Sykes (*Oxford*).

Session 5: New Concepts in Respiratory Care. Chairman: Professor J.G. Jones. A new model for the alveolus, Professor B.A. Hills (*New South Wales*). Nocturnal sleep and hypoxaemia in the first week after surgery, Professor R.L. Knill (*Western Ontario*). Asthma and the anaesthetist, Dr G.M. Cochrane (*Guy's Hospital*).

Session 6: Anaesthesia and Surgery Enquiry. Chairman: Dr T.B. Boulton. Confidential enquiry into perioperative deaths: an interim report, Dr J.N. Lunn (*Cardiff*).

Session 7: Free Papers. Chairman: Dr R.M. Jones. New blood-gas variables for the rational control of oxygen therapy, Dr M.C.C. Clapham, Professor W.W. Mapleson, Mr N. Willis (*University of Wales College of Medicine, Cardiff*). Acupuncture minimises opioid-induced vomiting and nausea, Dr A.G.A. Lynas, Professor J.W. Dundee, Dr R.G. Ghaly (*Belfast*). Clinical pharmacology of BW B1090U continuous infusion, Dr H.H. Ali, Dr J.J. Savarese, Dr P.B. Embree, Dr S.J. Basta, Dr L.H. Bottros, Dr J.N. Weakly, Dr A.G. Batson (*Boston, USA*). Comparison of BW A938U and pancuronium in anaesthetised patients, Dr M.P. Mehta, Dr D. Murray, Dr R. Forbes, Dr W.W. Choi, Dr S.D. Gergis, Dr M.D. Sokoll, Dr M.M. Abou-Donia, Dr G.D. Rudd (*Iowa, USA*).

Session 8: Free Papers. Chairman: Dr R.P. Wise. Predicting outcome from critical illness; applications of sickness scoring, Dr J.F. Bion, Dr T.C. Aitchison, Professor S.A. Edlin, Professor I. McA. Ledingham

(*Glasgow and Plymouth*). The use of protocols in obstetric units, Dr B. Owen (*Rhyl*). Guidelines for pre-anaesthetic investigation – blood ordering – outpatient assessment. Constructive clinical budgeting, Dr J.M. Cundy, Dr M. Hatton (*Lewisham*). Nitrogen washout from the circle absorber system during low flow anaesthesia, Dr A.A. Tomlinson, Dr P.J. Horsey, Dr H. Crooks (*Stoke-on-Trent and Southampton*).

The John Snow Memorial Lecture was given by Professor A.W. Clare (*St Bartholomew's Hospital*).

Poster presentations: Transarterial brachial plexus block using 50 ml of 1.5% mepivacaine, Dr E. Cockings, Dr P. Moore (*Texas*). Anaesthesia experiences in developing countries, Dr S.I. Samuels, Dr J.B. Brodsky, Dr J. Wyner, Dr M. Champeau (*Stanford, California*). The effect of metoclopramide on the absorption of morphine from oral controlled release tablets, Dr A.R. Manara, Dr L. Mendel, Dr M.P. Shelly, Dr G.R. Park (*Cambridge*). Multiple functions of the airway intubator, Dr R. Tudor Williams (*Calgary*). H_2 antagonists and bupivacaine clearance, Dr G.M. O'Sullivan, Dr M. Smith, Dr B. Morgan, Dr F. Reynolds (*St Thomas's Hospital and Queen Charlotte's Maternity Hospital, London*). Children's drug dosages, Dr M.E. Stuart Taylor, Dr J.A. Lack (*Salisbury*). Prevention of regurgitation of stomach contents by a modified nasogastric tube with an integral balloon, Dr S.K. Lahiri (*Bristol*). Ninety- and 120-second tracheal intubation with BW B1090U: clinical conditions with and without priming after fentanyl–thiopental induction, Dr J.J. Saverese, Dr H.D. Ali, Dr S.J. Basta, Dr P.B. Embree, Dr A. Schwartz, Dr L. Halleem-Bottros, Dr J.N. Weakly, Dr A.G. Batson (*Boston, USA*). Neuromuscular and cardiovascular effects in patients of BW A938U: a new long-acting neuromuscular blocking agent, Dr S.J. Basta, Dr J.J. Savarese, Dr N. Sunder, Dr L.H. Bottross, Dr H.H. Ali, Dr P. Embree, Dr A. Schwartz, Dr F. Varin, Dr G.D. Rudd, Dr J.N. Weakly (*Boston, USA*).

Future meetings of the Association

Annual Scientific meetings

1987—Sheffield, 10–11 September.
1988—Southampton, 15–16 September.
1989—Swansea, 14–15 September.

Linkman conferences

1987—Sheffield, 9 September.
1988—Southampton, 14 September.
1989—Swansea, 13 September.

Other meetings

1987—London, 17 January. Symposium on Management for Anaesthetists.
1987—London, 20–23 July. 2nd International Symposium on the History of Anaesthesia.

Membership subscriptions

Direct Debit

Members resident in the United Kingdom are reminded of the advantages of the Direct Debit method of payment. Annual subscriptions can only be increased by resolution at the Annual General Meeting and members must be informed of any increase. Direct Debit forms can be obtained from the Honorary Treasurer. *Overseas members*, unless they have a United Kingdom bank account, should pay by cheque or Banker's draft. Members not wishing to use the Direct Debit system should pay by cheque.

Research grants

Research grants may be made towards a salary for a doctor or technician, or for the cost of equipment. Candidates should write for full information to the Honorary Secretary and applications should be made on the special form designed for this purpose. Grants are unlikely to exceed £5000 per annum to an individual or department. Applications may be submitted any time during the year for consideration at the next meeting of the Research and Education Committee. This committee meets three times each year.

Association travel grants

Association travel grants are available towards the cost of a tour, either at home or abroad, including teaching research or study, but neither for the purposes of attending meetings of societies nor for taking up posts abroad. Grants of up to £500 may be awarded.

Applications should be made on a special form which may be obtained from the Honorary Secretary and may be submitted at any time during the year, provided sufficient time is allowed for their consideration by the Research and Education Committee.

Awards for educational visits to third world countries

Council has recently created an additional award for those who are making relatively prolonged visits, normally of not less than one year, to countries to assist with the teaching of anaesthesia. These awards would offer sums of up to £1000 towards expenses incurred.

Applications should be made to the Honorary Secretary and may be submitted at any time of the year.

Association Travenol Travelling Fellowship

Travenol Laboratories have endowed a travelling fellowship to the value of £2500, which will be awarded on an annual basis and is open to all members of the Association of Anaesthetists. The award may be used for travel within the United Kingdom or overseas to undertake a hospital-based study to examine current problems, new ideas, fresh applications or alternative systems in the following fields: intravenous fluid therapy, parenteral nutrition, intravenous administration of drugs, blood and blood component therapy, intravenous solution and transfusion fluid filtration, or education and training in regard to any of these.

Applications for the 1987 award, which should enclose a summary of at least 1000 words giving details of the objectives of the proposed study, should be sent to the Honorary Secretary from whom further information may be obtained, before 1 June 1987.

Fees for anaesthetists in independent practice in the United Kingdom

Guidelines on the scale of fees which should be charged in independent practice have been prepared by the Association and revised January 1986. Copies of this information can be obtained from the Honorary Secretary.

Central catalogue of historical books and equipment

A central national catalogue of the location of historical anaesthetic equipment and books is being prepared by the Association Archivist, Dr T.B. Boulton (*Oxford and Reading*). Dr D. Wilkinson (*St Bartholomew's Hospital*) is preparing a catalogue of apparatus and Dr I. McLellan (*Leicester*) is preparing the catalogue of books. The Honorary Secretary would be grateful to receive lists of historical equipment and books from members who have them in their charge.

Publications of the Association of Anaesthetists of Great Britain and Ireland

Your anaesthetic (some questions answered), an explanatory leaflet for patients, 1986. *Anaesthetic record cards*, suitable for independent medical practice, 1978. *The role of the anaesthetist*, an exploratory study by Miss Lisl Klein, 1980. *Report of a Working Party on the use of controlled drugs in operating theatres*, 1982. *Report of a Working Party on assistance for anaesthetists*, 1983. *Staffing in a District General Hospital*, 1983. *Workload for consultant anaesthetists* (revised), 1983. *Fees in independent practice*, 1986. *Workload for consultant anaesthetists*, implications of reductions in junior staff and the consultant-only hospital, 1984. *Anaesthetic service in isolated units in the United Kingdom* (prepared jointly with the Anaesthetists' Sub-Committee of CCHMS), 1984. *Consultant Advisory Appointments Committees*, results of a questionnaire, 1984. *Scavenging in theatres*, results of a questionnaire, 1984. *Anaesthesia, a career guide*, an illustrated booklet designed to inform senior medical students and preregistration house officers of the value of postgraduate training in anaesthesia (recently revised to include information on the new examination system), 1984. *Professional References in the National Health Service*, guidance and proforma, 1985. *Post-Anaesthetic Recovery Facilities*, 1985. These are available from the Association offices.

'Near miss' reporting

Council of the Association intends to publish, from time to time, instructive examples of 'near-misses' which have educational value whether or not they led to any serious morbidity or mortality. Readers are invited to submit accounts of the genesis, detection and correction of incidents, giving sufficient detail as to make the matter intelligible to practising anaesthetists and to point out the educational aspect.

Reports should generally be between 150–500 words in length but submissions may be longer than this and will be edited if necessary. They will be published anonymously.

Editorial work will be undertaken after the secretariat has removed any trace of personal identification. The style aimed for will be on the lines of clinical incidents currently published by the defence societies. Please submit any incidents you think worthy of report to the Honorary Secretary, Critical Incident Reports, at the Association offices.

Undergraduate prize of the Association

Winners 1986

1st prize: Mr S.K. Hogan (*Leeds*), The management of postoperative pain. Room for improvement? *2nd prize*: Mr G. Matfin (*Leeds*), Infection in the neonatal intensive care unit. *3rd prize*: Ms Deborah A. Wales (*Leeds*), A letter to Mendelson.

Undergraduate prize—1987

The Prizes are offered for the most meritorious essays written by an undergraduate medical student at a university in Great Britain or Ireland. The competition is in two classes with prizes in each class of £100, £50 and £25.

Class 1—aspects of basic science of anaesthesia.
Class 2—clinical aspects of anaesthesia.

Rules

Essays should be submitted before 1 June 1987. The number of entrants from any one medical school is limited to a maximum of five. The Association recognises that most medical schools already offer prizes to medical students for an essay on a topic related to the specialty and has therefore decided that the winning of a local prize will not preclude the writer from entering for the Association prize.

Essays should be prepared according to the general format of the 'Notice to Contributors' at the end of each issue of *Anaesthesia*. Three copies of the essay should be forwarded to the Honorary Secretary.

Regional and Specialist Society Meeting Dates

The Honorary Secretary once again appeals to the secretaries of regional and specialist anaesthetic societies to try to check that their proposed meetings do not conflict with other previously arranged events to their mutual detriment. Dr A. Padfield, Royal Hallamshire Hospital, Sheffield S10 2RX, sub-editor of the National and local events section, has advance notice of meeting dates beyond those published in that section.

Competence to practise

The Association of Anaesthetists of Great Britain and Ireland and the Royal College of Psychiatrists are cooperating in a scheme to help sick anaesthetists early in their illness.

The system depends upon voluntary reporting by colleagues and is not coercive. It must be emphasised that this scheme is supplementary to existing controls. Fitness to practise may be impaired through illness and the sick doctor for various reasons may not take any steps to remedy the situation. The scheme provides machinery whereby confidential psychiatric help can be offered to the ailing doctor from a source outside his own Region.

The reporting clinician should contact the office of the Association of Anaesthetists to ascertain the name of a referee (Dr P.J. Helliwell, Dr D.C. Howat, Dr S.A. Mason, Dr D.B. Scott) and need not identify himself to the office staff. The reporting clinician will be given the name, telephone number and availability of the referee. Preferably, a second clinician will also be required to confirm the report to the referee. The referee will then contact a psychiatrist from another Region nominated by the College of Psychiatrists from a prepared list: the psychiatrist will then get in touch with the potential patient.

No further information will be transmitted through the system unless the potential patient does not accept the offer of a visit to the nominated psychiatrist. In that case, the reporting physician(s) will be informed after a month that treatment has not been accepted.

Suitable arrangements have been made for confidential treatment of the doctor–patient in another Region and for the reimbursement of travel expenses.

Other relevant publications

WFSA Cardiopulmonary Cerebral Resuscitation Manual. Those members wishing to receive a copy should send a stamped addressed envelope measuring not less than 175 × 250 mm and bearing stamps to the value of £1 (United Kingdom) to Dr P.J.F. Baskett, Secretary/Treasurer, Cardiopulmonary Resuscitation Committee WFSA, at the Association offices.

WFSA Manual on Obstetric Analgesia and Anaesthesia. Copies of this manual are available to members for £2 plus 75p postage. Please send a stamp and addressed envelope to the Honorary Secretary at the Association offices.

Mortality associated with anaesthesia. The report on the Association's study was published by the Nuffield Provincial Hospital Trust (3 Prince Albert Road, London NW1 7SP) from whom copies may be purchased. Members of the Association who wish to receive a copy should send £1 (to cover postage and administrative charges) to the Association offices with their request.

Guidelines for the basic facilities and equipment required for the administration of anaesthetics in hospitals in the European Economic Community, published by the UEMS (Anaesthesia and Intensive Care Committee). Photocopies are available from the Association offices.

Ties

Ties (multiple motif) are available in twinzine/terylene in navy, maroon or green, at £4.50 each, post free.

A new design of tie, which depicts the Coat of Arms of the Association, is now available in pure silk (navy only) at £7.50 each, post free or terylene (navy, maroon or green) at £5.00 each post free (inland postage). Orders including your cheque, to the Association office.

Catalogues and slides of the Charles King collection of historical apparatus

The Development of Anaesthetic Apparatus. A History based on the Charles King Collection. Price: £27.50 (£19.50 to Members of the Association of Anaesthetists or subscribers to *Anaesthesia*). Post and packing £1.75.

Colour Slides of the Charles King Collection. 8 sets of 12 slides. List on request. Prices: One set £5 (p & p 59p). 8 sets £37.50 (p & p £1.75). 8 sets plus the book *Development of Anaesthetic Apparatus* £55 (p & p £2.35).

These items can be ordered from the Association offices.

Souvenirs of the Golden Jubilee of the Foundation of the Association of Anaesthetists of Great Britain and Ireland

The following commemorative souvenirs can be obtained by writing to the Association offices. A few of the following remain and can be obtained at reduced prices.

Wall plaque of the Arms of the Association of Great Britain and Ireland in full heraldic colours (18 × 15 cm) £5.00
Wall plaque of the badge of the World Federation of Societies of Anaesthesiologists (WFSA) £5.00
Lead crystal glass tankard (0.6 litre) engraved with the Arms of the Association and inscribed 'Golden Jubilee 1932–1982' (limited edition of 250, in a presentation box) £7.50
Fine bone china plate, by Wedgwood. Marked limited edition of 250. Diameter 23 cm. Association Coat of Arms (gold with red and blue lettering on a white background, in a presentation box) £15.00

Audiovisual material for sale or hire

The following audiovisual material is the property of the Association and may be bought or hired.

16-mm films

'Lest we forget—1'. Four brief films made in conjunction with the Section of Anaesthetics of the Royal Society of Medicine, which record a meeting held in December 1974 under the Presidency of the late Dr C.J. Massey Dawkins. (Blind nasal intubation, Sir I.W. Magill; Spinal analgesia, Sir R.R. Macintosh; Cyclopropane, Dr M.D. Nosworthy; Vinyl ether, Dr W. McConnell).

These films may be hired at the nominal charge of £8.50 each + postage. Copies can be ordered for purchase and quotations are available on enquiry.

Audiocassettes

These are for sale only. All four talks, from each meeting are on one cassette and no slides were used (£3.00).

'Lest we forget—1'. This is a sound recording of the previously described meeting.

'Lest we forget—2'. This is a sound recording of a second meeting held under the Presidency of Dr C.F. Scurr in March 1979. The contributors were: Sir G.S.W. Organe (The introduction of muscle relaxants), Professor A.C. Forrester (Hypothermia), Dr A.I.P. Brown (Flat on one's face) and Dr J.A. Lee (Spinal analgesia).

The Junior Anaesthetists' Group of the Association of Anaesthetists of Great Britain and Ireland

All junior members of the Association are members of the Group. The Group committee has representation on various committees of the Association and Faculty.

Chairman: Dr G.W. Hamlin (*Leeds*).
Honorary Secretary: Dr L. Allan (*51, Salisbury Road, Newington, Edinburgh*).

Future meetings

1987—8–10 April. Belfast. (Junior Linkman Meeting, April 7).
1988—5–8 April. Nottingham.

The Junior Anaesthetists' Annual Prize Competition

This is a competition held at the Annual Scientific Meeting of the Junior Anaesthetists' Group (Belfast, 8–10 April 1987) and consists of 10-minute papers which are presented by junior members at a special session of the meeting. The rules for this competition were published in *Anaesthesia* 1982; **37:** 129–30.

The closing date for submission of abstracts for this competition is 1 February 1987, and entries and abstracts should be forwarded by then to the Association offices.

Faculty of Anaesthetists of the Royal College of Surgeons of England

The Faculty of Anaesthetists of the Royal College of Surgeons of England, 35–43 Lincoln's Inn Fields, London WC2A 3PN. Telephone: 01–405 3474.

Nuffield Prize

The Nuffield Prize was awarded to Dr A.J. Hill on the results of the Part II FFARCS examination held in April and May 1986.

Fellowship Prize

The Fellowship Prize was awarded to Dr Rhys David Evans on the results of the Part III FFARCS examination held in July 1986.

Examination results

At the Part II Fellowship examinations held in October 1986 125 of the 287 candidates were successful.

Future meetings

1987—18 March. Anniversary Forum. Paediatric anaesthesia in a District General Hospital.
—8 May. Scientific meeting. Anaesthesia and myocardial ischaemia.

Faculty of Anaesthetists of the Royal College of Surgeons in Ireland

The Faculty of Anaesthetists of the Royal College of Surgeons in Ireland, 123 St Stephen's Green, Dublin 2.
Telephone: Dublin 780200 (Ext 268).

Dean: Dr P.W. Keane.
Vice-Dean: Dr D.C. Moriarty.
Honorary Secretary: Dr D.F. Hogan.
Honorary Treasurer: Dr J.C. Cooper.

Educational activities

Annual Scientific Meeting of the Faculty. 15–16 May 1987.

Section of Anaesthetics of the Royal Society of Medicine

Royal Society of Medicine, 1 Wimpole Street, London W1M 8AE. Telephone: 01–580 2070.

President: Dr W.D.A. Smith, OBE.
Secretaries: Dr G.M.C. Paterson (*Nuffield Department of Anaesthetics, The Radcliffe Infirmary, Oxford OX2 6HE*), Professor T.E.J. Healy (*Manchester*).

Meetings of the Section are held on the first Friday in the month, beginning in November, and are held at 1 Wimpole Street, London W1M 8AE, at 5.30 p.m.

Fellows of the RSM are welcome to bring guests if they wish.

Programme

1987

9 January—*Work in progress: applications of spinal anaesthesia.*
6 February—*Symposium on legal aspects of anaesthesia.*
6 March—*Practice makes perfect?—Studies on repeat anaesthetics.*
3 April—*Anaesthesia and surgery for congenital craniofacial abnormalities.*
8 May—*May & Baker R.S.M. Prizes, AGM, Annual Dinner.*
5 June—*All-day symposium: medical emergencies.*
2 October—*Sir Robert Macintosh's 90th Birthday (17 October 1987): a tribute. Followed by a reception given jointly with the Association of Anaesthetists of Great Britain and Ireland and the Faculty of Anaesthetists of the Royal College of Surgeons of England.*

May and Baker R.S.M. Prizes

The President and the Council earnestly request Members of the Section to draw the attention of their junior staff to the May and Baker R.S.M. Prizes (Anaesthetics). Copies of the regulations may be obtained from the Sections Office, 1 Wimpole Street, London W1M 8AE.

Specialist Societies

British Association for Immediate Care (BASIC)

Chairman: Dr H. MacLeod.
Hon. Secretary: Dr Judith Fisher.
Administrator: Mr R. Bailey, BASIC (*31c Lower Brook Street, Ipswich IP4*).

Society of Anaesthetists of the British Forces in Germany

President: Dr P.J.F. Baskett (*Bristol*).
Honorary Secretary: Squadron Leader M.J. Wort (*R.A.F. Hospital, Wegberg, BFPO 20, Germany*).

Association of Cardiothoracic Anaesthetists

Honorary Secretary: Dr R.D. Latimer (*Papworth Hospital, Papworth Everard, Cambridge CB3 8RE. Tel: 0480 830541*).

Future meetings

1987—26 June. Cambridge.
Information: Dr I. Hardy (*Papworth Hospital, Papworth Everard, Cambridge CB3 8RE*).
—25 September. Liverpool.
Information: Dr J.C. Richardson (*Broadgreen Hospital, Thomas Drive, Liverpool L14 3LB*).
1988—Spring Meeting. Cambridge.
Information: Dr J. Kneeshaw (*Papworth Hospital, Papworth Everard, Cambridge CB3 8RE*).

Association of Dental Anaesthetists

President: Dr T.M. Young (*Manchester*).
Secretary: Dr M.A. Skivington (*Department of Anaesthetics, Poole General Hospital, Poole, Dorset BH15 2JB*).
Assistant Secretary/Editor: Dr M.E. Ward (*Oxford*).

Future meetings

1987—17 January. Guy's Hospital, London (please note change of venue).
Information: Dr R.M. Jones (*Department of Anaesthetics, Guy's Hospital, London SE1 9RT. Tel: 01–407 7600*).
—3–4 July. Plymouth.
Information: Dr F.S. Keddie.
1988—1–4 June. Guernsey.

European Association of Cardiothoracic Anaesthetists

Honorary Secretary/Treasurer: Dr R. Latimer (*Papworth Hospital, Papworth Everard, Cambridge CB3 8RE. Tel: 0480 830541*).

Future meeting

1987—19–21 May. Gottingen, West Germany.
Information: Professor D. Kettler (*Georg August University, Robert Koch Strasse 40, D-3400, West Germany*).

History of Anaesthesia Society

President: Dr J.A. Lee (*Southend-on-Sea*).
Vice-President: Dr T.B. Boulton (*Oxford and Reading*).
Honorary Treasurer: Dr A. Padfield (*Sheffield*).
Honorary Secretary: Dr I. McLellan (*Department of Anaesthesia, Groby Road Hospital, Leicester LE3 9QE*).

Membership

Membership is open to all those interested in the history of anaesthesia, whether medically qualified or not.

Future meeting

1987—7 February. St Bartholomew's Hospital, London. The host will be Dr D. Wilkinson. Free papers are invited.
Information: Dr D. Wilkinson (*St Bartholomew's Hospital, London EC1A 7BE*).

Intensive Care Society

Chairman: Dr M.A. Branthwaite (*Brompton*).
Meetings Secretary: Dr E. Major (*Swansea*).
Secretary/Treasurer: Dr D.W. Ryan (*Intensive Therapy Unit, Freeman Hospital, Newcastle-upon-Tyne NE7 7BE*).

Future meeting

1987—9–11 April, Exeter University.
Information: Dr C.J. Hinds.

The Intractable Pain Society of Great Britain and Ireland

Chairman: Dr K. Budd (*Bradford*).
Honorary Secretary: Dr T.P. Nash (*Basingstoke District Hospital, Aldermaston Road, Basingstoke, Hampshire RG24 9NA*).

Prize Essay

An annual competition is held for the best essays related to pain relief. Submissions to the Honorary Secretary by 31 August 1987. Essay contents are judged in two groups, Doctors in training and Society members.

Future meetings

1987—10–11 April. Annual General Meeting and Scientific Meeting. Liverpool.
Hosts: Drs C. Wells and S. Lipton.
—5–6 June. Behavioural treatments in chronic pain. A celebration of 150th anniversary of St Andrew's Hospital, Northampton.
Host: Dr K. Trick.

Neuroanaesthetists' Travelling Club

President: Dr D. Powell (*Leeds*).
Honorary Secretary: Dr J.L. Jenkinson (*Department of Surgical Neurology, The Western General Hospital, Crewe Road, Edinburgh EH4 2XU*).

Future meetings

1987—22–25 March. Joint meeting with German Anaesthetists. Tubingen.
Information: Honorary Secretary.
—9 May. Annual meeting. Leeds.
McDowall Lecture: Professor J.D. Michenfelder, MD, Mayo Medical School.
Information: Dr D. Powell (*The General Infirmary at Leeds, Great George Street, Leeds LS1 3EX*).

1988—14–16 April. Joint meeting with the Society of British Neurosurgeons, and Annual Meeting. Oxford.
Information: Dr J. Edmonds-Seal (*Nuffield Department of Anaesthetics, The Radcliffe Infirmary, Oxford OX2 6HE*).
—20–22 May. Joint meeting with the American Society of Neurosurgical Anaesthesia and Neurologic Supportive Care. Williamsburg.
Information: Honorary Secretary.

Obstetric Anaesthetists' Association

President: Dr D.D. Moir (*Glasgow*).
Honorary Secretary: Dr J. Thorburn (*Department of Anaesthesia, The Queen Mother's Hospital, Yorkhill, Glasgow G3 85H*).
Treasurer: Dr A.P. Rubin (*London*).
Minute Secretary: Dr B.R. Wilkey (*Cambridge*).
Committee: Professor M. Rosen, Drs R.G. MacDonald, B. Owen, M. Milne, F. Russell, A. Singh, J. Wilson, J. Moore, M. Tunstall, L.E.S. Carrie.

All UK membership enquiries to Dr J. Thorburn please.

Autumn meeting

This was held in Jordanstown, University of Ulster, on 5 September, 1986. Fifteen papers were delivered, as follows. Place and method of high risk fetus, Dr J. Dorman. Who's baby is it anyway?, Dr M.A. Lewis. Organisation of resuscitation services, Dr G. McClure. Epidural or spinal anaesthesia for operative delivery, Prof. M. Finster. Predictability and blood pressure in spinal anaesthesia for Caesarean section, Dr M. Tunstall. Spinal anaesthesia for elective Caesarean section, comparisons of bupivacaine and cinchocaine, Dr R.N. Freeman. Cardiac disease in pregnancy, Dr P. Morton. Anaesthesia and the pregnant cardiac patient, Dr D.D. Wilson. Epidural anaesthesia survey on obstetrics in Northern Ireland, Dr J.C. Moffett. Evolution of epidural service in a District hospital, Dr I.M. Bali. Audit of an epidural service at a District General hospital, Dr H.E. Hosie. A new system for epidural top ups, Dr G. Russell. Not a laughing matter, Dr C.R.D. Laird. Phenytoin infusions in the treatment of severe pre-eclampsia, Dr R.M. Slater. Anaesthetist's role in the case of a severely pre-eclamptic mother, Dr J.S. Crawford.

Future meeting

1987—10 April, Hull.
Information: Dr I.F. Russell (*Westwood Hospital, Beverley, North Humberside*).

Association of Paediatric Anaesthetists

President: Dr G.W. Black (*Belfast*).
Honorary Secretary: Dr P. Morris (*Royal Manchester Children's Hospital, Pendlebury, Manchester M27 1HA*).

Future meetings

1987—6–7 March. Annual Meeting. Leeds.
Free paper submissions (with synopsis) for this meeting should be made to the Secretary as soon as possible after 1 January 1987.
Further information: Honorary Secretary.
—12–13 June. Great Ormond Street, London.
3rd APA seminar on paediatric anaesthesia.
Information: Dr D.J. Hatch (*Department of Anaesthesia, The Hospital for Sick Children, Great Ormond Street, London WC1N 3JH*).
—2–3 October. Joint meeting with the French Society of Paediatric Anaesthetists in Paris (please note change of date of this meeting).
Information: Dr W.R. Hain (*Children's Intensive Care Unit, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH*).

Plastic Surgery and Burns Anaesthetists

President: Dr R.D. Jack (*Wexham Park*).
Honorary Secretary: Dr F.J.M. Walters (*Department of Anaesthetics, Frenchay Hospital, Bristol BS16 1LE*).

Enquiries about membership to the Honorary Secretary please.

Autumn meeting

The following papers were presented. Surgical developments for cleft lip and palate surgery, Mr R.W. Piggott (*Frenchay*). Anaesthetic developments for cleft lip and palate surgery, Dr F.J.M. Walters (*Frenchay*). Management of multiple burn injuries, Mr R. Griffiths (*Sheffield*). Surgical aspects of craniofacial deformities, Mr D. Soutar (*Canniesburn*). Anaesthesia for craniofacial surgery, Dr V. Goat (*Oxford*). Anaesthesia for microsurgery, Dr E. Whelan (*Whiston*). A new vasoconstrictor for plastic surgery, Dr V. Capouet (*Brussels*). The use of Emla cream in children, Dr C.S. Hopkins (*Liverpool*). A new device for oxygenation during difficult tracheal intubation, Drs M.E. Ward and V. Goat (*Oxford*).

Future meeting

1987—26 September. Manchester.
Information: Dr C. Tolhurst-Cleaver (*University Hospital of Manchester. Tel. 061–445 8111*).

Anaesthetic Research Society

Chairman: Professor A.A. Spence.
Honorary Secretary: Dr J.W. Sear (*Nuffield Department of Anaesthetics, John Radcliffe Hospital, Oxford OX3 9DU*).

Future meetings (closing dates for papers)

1987—20 March (6 February). University of Oxford.
—26–27 June (8 May). University of Glasgow.
—13–14 November (25 September). Northwick Park Hospital.

Special Notice

Abstracts are photoreduced and must be submitted on a preprinted form, which is available from the Honorary Secretary. All correspondence and abstracts should be sent to the Honorary Secretary.

Resuscitation Council (UK)

Chairman: Mr A. Marsden.
Secretary: Dr D.A. Zideman (*Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 OHS*).

Advanced Life Support

Posters which illustrate the Council's recommendations for Advanced Life Support are now available.

Home news and notices

SmithKline Beckman Professor

Professor C. Prys Roberts (*Bristol*) was appointed to one of these visiting posts to the USA for 1986.

A new chair

The British Oxygen Company Group has recently announced that it is to fund a new BOC chair in the Department of Anaesthetics at the Royal College of Surgeons of England.

National and local events

The diary is collated by Dr A. Padfield, Consultant Anaesthetist, Royal Hallamshire Hospital, Sheffield S10 2RX. Society secretaries are invited to submit their programmes to him.

January 1987

7 *Northern Ireland Society of Anaesthetists.* Dr R.M. Jones. *Perioperative cardiac dysrhythmias.* *Information:* Dr J.E. Hegarty, Department of Anaesthetics, 97 Lisburn Rd, Belfast BT9 7BL.

9 *FFA Part 3.* London. Written papers.

9 *Section of Anaesthetics, Royal Society of Medicine. Work in progress: Spinal anaesthesia.* *Information:* Sections Office, RSM, 1 Wimpole St, London W1M 8AE.

13 *FFA Part 1/DA(UK).* London. Applications due.

13 *Reading Anaesthetists.* Mr N. Marks. *Snoring.* *Information:* Dr R. Marshall Barr, Royal Berkshire Hospital, Reading, Berks. RG1 5AN.

14 *Yorkshire Society of Anaesthetists.* Presentations from the University Department. *Information:* Dr J.S. Gibson, Leeds General Infirmary, Leeds LS1 3EX.

15 *Manchester Medical Society, Section of Anaesthetics.* Professor A. Percival (Bacteriology). *Information:* Dr E.E.M. Thompson, University of Manchester, Oxford Rd, Manchester M13 9PL.

16 *Liverpool Society of Anaesthetists.* Presentations by the University Department. *Information:* Dr Anne M. Florence, Broadgreen Hospital, Liverpool L14 3LB.

17 *Association of Dental Anaesthetists.* Guy's Hospital, London. *Host:* Dr R.M. Jones, Department of Anaesthetics, Guy's Hospital, London SE1 9RT.

24 *Glasgow and West of Scotland Society of Anaesthetists.* Members' Night: Ayrshire Division. *Information:* Dr Margaret Stockwell, Division of Anaesthetics, Glasgow Royal Infirmary, Glasgow G31 1ER.

February 1987

4 *Northern Ireland Society.* Professor I. McA. Ledingham. *The role of intensive care in modern medicine.*

Information: Dr J.E. Hegarty, Department of Anaesthetics, 97 Lisburn Rd, Belfast BT9 7BL.

6 *Section of Anaesthetics, Royal Society of Medicine. Symposium: legal aspects of anaesthesia.* *Information:* Sections Office, RSM, 1 Wimpole St, London W1M 8AE.

7 *History of Anaesthesia Society.* St Bartholomew's Hospital. *Information:* Dr D.J. Wilkinson, Department of Anaesthetics, St Bartholomew's Hospital, London E34.

9 *FFA Part 3.* London. Vivas begin.

11 *Reading Anaesthetists.* Dr C. Waldmann. *Anaesthesia at altitude and depth.* *Information:* Dr A.M. Barr, Royal Berkshire Hospital, Reading, Berks. RG1 5AN.

12 *Yorkshire Society.* Annual General Meeting. *Presidential address:* Dr P.K. Storah. *Information:* Dr J.S. Gibson, Leeds General Infirmary, Leeds LS1 3EX.

19 *Manchester Medical Society, Section of Anaesthetics.* ICI & Abbott Registrars' Prizes/Annual General Meeting. *Information:* Dr E.E.M. Thompson, University of Manchester, Oxford Rd, Manchester M13 9PL.

20 *Liverpool Society.* Registrars' Prize Papers. *Information:* Dr A.M. Florence, Broadgreen Hospital, Liverpool L14 3LB.

23 *Wessex Anaesthetic Club.* Dr C.D. Hanning. *'He looks a little blue at my end'.* *Information:* Dr D.V. Rutter, Level E, Centre Block, Southampton General Hospital, Southampton SO9 4XY.

24 *FFA Part 1/DA(UK).* London. Written papers.

24 *Glasgow and West of Scotland Society.* Dr M. Halsey. *Clinical implications of the mechanisms of anaesthesia.* *Information:* Dr M. Stockwell, Division of Anaesthetics, Glasgow Royal Infirmary, Glasgow G31 1ER.

26 *Study day in anaesthesia for neurosurgery and neurological diseases.* *Information:* Mrs W. Scott, West of Scotland Committee for Postgraduate Medical Education, University of Glasgow, Glasgow G12 8QQ.

March 1987

6 *Section of Anaesthetics, Royal Society of Medicine. Practice makes perfect?—Studies on repeat anaesthetics from Oxford.* *Information:* Sections Office, RSM, 1 Wimpole St, London W1M 8AE.

6–7 *Association of Paediatric Anaesthetists.* Leeds. *Information:* Dr P. Morris, Royal Manchester Children's Hospital, Manchester M27 1HA.

10 *Reading Anaesthetists.* Dr A. Boon. *Aspects of neonatal ventilation.* *Information:* Dr A. Marshall Barr, Royal Berkshire Hospital, Reading, Berks. RG1 5AN.

11 *Northern Ireland Society of Anaesthetists.* Professor J.W. Dundee. *No man is an island.* *Information:* Dr J.E. Hegarty, Department of Anaesthetics, 97 Lisburn Road, Belfast BT9 7BL.

13 *Guy's Anaesthetic Symposium. Safety in anaesthesia.* *Information:* Dr N.I. Newton, Guy's Hospital, London SE1 9RT

14 *Society of Anaesthetists of Wales.* Spring Scientific Meeting. Talygarn. *Information:* Dr R. Mason, Department of Anaesthetics, Morriston Hospital, Swansea SA6 6NL.

18 *FFA Part 2.* London. Applications due.

18 *Faculty Anniversary Forum. Paediatric anaesthesia in the District General Hospital.* *Information:* Faculty of Anaesthetists, 35/43 Lincoln's Inn Fields, London WC2A 3PN.

20 *Anaesthetic Research Society.* Oxford. *Information:* Dr A.R. Aitkenhead, Leicester Royal Infirmary, Leicester LE1 5WW.

20 *North of England Society of Anaesthetists.* Title to be announced. *Information:* Dr J.E. Charlton, Royal Victoria Infirmary, Newcastle-upon-Tyne NE1 4LP.

23 *FFA Part 1/DA(UK).* London. Vivas begin.

25 *Glasgow and West of Scotland Society of Anaesthetists. Presidential address:* Dr W.L.M. Baird. *Information:* Dr M. Stockwell, Division of Anaesthetics, Glasgow Royal Infirmary, Glasgow G31 1ER.

26 *Northern Ireland Society of Anaesthetists.* Annual Dinner. *Information:* Dr J.E. Hegarty, Department of Anaesthetics, 97 Lisburn Road, Belfast BT9 7BL.

27 *Section of Anaesthetics, Manchester Medical Society.* Joint meeting with the *Liverpool Society.* Liverpool. *Speakers:* Dr M.M. Burrows, Dr N.J.N. Harper. *Information:* Dr E.E.M. Thompson, University of Manchester, Oxford Rd, Manchester M13 9PL.

April 1987

2–3 *West Midlands Intensive Care Group.* 7th Annual Meeting. Warwick University. *Information:* Dr T.J. Digger, Russells Hall Hospital, Dudley, West Midlands DY1 2HQ.

3 *Section of Anaesthetics, Royal Society of Medicine. Anaesthesia and surgery for congenital cranio-facial abnormalities,* from the Oxford Cranio-facial Unit. *Information:* Sections Office, RSM, 1 Wimpole St, London W1M 8AE.

7–10 *Junior Anaesthetists' Group.* Linkman, Annual

General Meeting and Annual Scientific Meeting. *Belfast.*
Information: Association of Anaesthetists, 9 Bedford Square, London WC1B 3RA.

9–11 *Intensive Care Society.* Spring Meeting. Exeter University.
Information: Dr C.J. Hinds, St Bartholomew's Hospital, London EC1A 7BE.

10 *Obstetric Anaesthetists' Association.* Hull.
Information: Dr J. Thorburn, Queen Mother's Hospital, Yorkhill, Glasgow G3 8SH.

14 *Reading Anaesthetists.* Dr J. McDonald. *Mobile intensive care.*
Information: Dr A. Marshall Barr, Royal Berkshire Hospital, Reading, Berks. RG1 5AN.

15 *Northern Ireland Society of Anaesthetists.* Annual General Meeting and Dr R.K. Mirakhur: *Monitoring and antagonism of neuromuscular block.*
Information: Dr J.E. Hegarty, Department of Anaesthetics, 97 Lisburn Road, Belfast BT9 7BL.

16 *FFA Part 3.* London. Applications due.

16 *Manchester Medical Society, Section of Anaesthetics.* Out of town meeting at Oldham Royal Infirmary. Dr J.A.W. Wildsmith: *Choices in regional anaesthesia.*
Information: Dr E.E.M. Thompson, University of Manchester, Oxford Rd, Manchester M13 9PL.

17 *North of England Society of Anaesthetists.* Symposium.
Information: Dr J.E. Charlton, Royal Victoria Infirmary, Newcastle-upon-Tyne NE1 4LP.

22 *Glasgow and West of Scotland Society of Anaesthetists.* Annual General Meeting.
Information: Dr M. Stockwell, Division of Anaesthetics, Glasgow Royal Infirmary, Glasgow G31 1ER.

24–26 *Scottish Society of Anaesthetists.* Annual Meeting. Peebles Hotel Hydro.
Information: Dr W.F.D. Hamilton, Department of Anaesthesia, Ninewells Hospital, Dundee DD1 9SY.

29 *FFARCS Part 2.* London. Papers.

International congress calendar

1987

12–15 January. Berlin. *10th National Congress 'Anaesthesia '87'.*
Information: Dr Pickart, Karowerstrasse 11, DDR-1115, Berlin-Buch, GDR.

17–24 January. Martinique, French West Indies. *Symposium on Advances in Anesthesiology, Clinical and Economic.*
Information: George Silvay, MD, PhD, Department of Anesthesiology, Mount Sinai Medical Centre, 1 Gustave L. Levy Place, New York 10029, USA. Tel: (212) 650 5954.

17–18 January. Washington, DC. *American Academy and Board of Head, Face and Neck Pain & TMJ Orthopedics.*
Information: American Academy and Board of Head Face and Neck Pain and TMJ Orthopedics, Medical Towers Building, Suite 803, 255 South 17th Street, Philadelphia, PA 19103, USA.

24–30 January. Beaver Run Resort, Breckenridge, Colorado. *Update on pediatric anaesthesia and intensive care.*
Information: UCHSC—Continuing Medical Education, 4200 East 9th Ave., Box C-295, Denver, CO 80262, USA.

31 January–7 February. Vail, Colorado. *12th Annual Vail Conference in Anesthesiology.*
Information: Professional Seminars, PO Box 012318, Miami, FL 33101, USA.

23–26 February. Sundial Beach & Tennis Resort, Sanibel Island, Florida. *Medical Management of the Surgical Patient.*
Information: Sandy Young, Program Coordinator, Office of Continuing Education, Turner 22, 720 Rutland Avenue, Baltimore, MD 21205, USA.

6–7 March. Basel, Switzerland. *7th Myron B. Laver International Postgraduate Course 'Anaesthesia and Intensive Care for the Neurosurgical Patient'.*
Information: Secretariat, 7th MBL IPGC, Department of Anaesthesia, University of Basel/Kantonsspital, CH-4031 Basel, Switzerland.

14–18 March. Florida, USA. *61st Congress of the International Anesthesia Research Society.*
Information: Emerson A. Moffitt, Executive Secretary, International Anesthesia Research Society, 3645 Warrensville Center Road, Cleveland, OH 44122, USA.

2–4 April. Tokyo. *34th Annual Meeting, Japan Society of Anesthesiologists.*
Information: Dr Kenjito Mori, 5F, TY Building, 18–11 Hongo 3-chome, Bunkyo-ku, Tokyo.

7–9 May. Paris. *VIth Annual Meeting of the European Society of Regional Anaesthesia.*
Information: M.A.P.A.R., Departement d'Anes-

thesiologie, Hopital de Bicetre, 94270 Kremlin-Bicetre, France.

19–21 May. Gottingen, W. Germany. *European Association of Cardiothoracic Anaesthetists.*
Information: Professor D. Kettler, Georg August Universitat, Robert Koch Strasse 40, D-3400, W. Germany.

28–31 May. Chania, Crete. *7th Congress of the Greek Society of Anaesthesiologists.*
Information: Greek Society of Anaesthesiologists, 34 Dragoumi Street, 115 28 Athens, Greece.

31 May–8 June. Paris, France. *International Seminar on Advances in Anesthesiology.*
Information: George Silvay, MD, PhD, Department of Anesthesiology, Mount Sinai Medical Centre, 1 Gustave L. Levy Place, New York 10029, USA. Tel: (212) 650 5954.

13–18 June. Calgary, Alberta. *Canadian Anaesthetists' Society Annual Meeting.*
Information: Mr Lloyd H. Mayeda, 94 Cumberland Street, Suite 901, Toronto, Ontario, Canada M5R 1AS.

20–25 June. Montreal, Canada. *XIIIth International Symposium on Cerebral Blood Flow and Metabolism.*
Information: Sorelcomm (1985) Inc., Conference Secretariat, 1425 Dorchester Boulevard West, Montreal, Canada H3G 1T2.

29 June–3 July. Linkoping. *19th Scandinavian Congress.*
Information: Department of Anaesthesiology, University Hospital, S-581 Linkoping, Sweden.

20–23 July. London. *2nd International Symposium on the History of Anaesthesia.*
Information: The Congress Secretariat, The Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1 3RA, UK.

2–7 August. Hamburg. *Vth World Congress on Pain.*
Information: Hamburg Messe & Congress, Postfach 30 24 80, 2000 Hamburg 36, FRG.

10 August. London. *Official Satellite Symposium of the I.A.S.A.P. Vth World Congress on Pain—Is Basic Research Helping Chronic Pain?*
Information: Dr D.M. Justins, PANG, Department of Anaesthesia, St Thomas's Hospital, London SE1 7EH, UK.

23–28 August. Sydney, Australia. *10th International Congress of Pharmacology.*
Information: The Secretariat, 10th IUPHAR Congress, G.P.O. Box 2609, Sydney, NSW 2001, Australia.

27–30 August. Kobe, Japan. *International Symposium on Cardiovascular Anesthesiology and 8th Annual Meeting of Japan Society of Circulation Control in Medicine.*
Information: Secretariat, ISCVA/SCC, Japan Convention Services Inc., Kansai Branch, Sumitomo Seimei Midosujui Building, 1 DF 4–14–3, Nishitemma, Kita-ku, Osaka 530, Japan.

7–9 September. Bali, Indonesia. *The 4th Congress of the Western Pacific Association of Critical Care Medicine (WPACCM).*
Information: Congress Secretariat, Indonesian Society for Critical Care Medicine (Perhimpunan Kedokteran Gawat Darurat Indonesia—PKGDI), P.O. Box 123/Jat.jg. Jakarta 13001, Jakarta, Indonesia.

10–11 September. Sheffield. *Annual Scientific Meeting, Association of Anaesthetists of Great Britain and Ireland.*
Information: Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA, UK.

15–18 September. Paris. *IIIrd International Symposium on Pediatric, Surgical and Neonatal Intensive Care.*
Information: S.O.C.F.I., 14 Rue Mandar, 75002 Paris, France.

10–14 October. Atlanta. *Annual Meeting, American Society of Anaesthesiologists.*
Information: John Andes, Esq., 515 Busse Highway, Park Ridge, IL 60068, USA.

22–24 October. Posnan, Poland. *Symposium–DYDAKTYKA '87.*
Information: The Organizing Committee of Symposium DYDAKTYKA '87, Institute of Anaesthesiology and Intensive Therapy, ul. Przybyszewskiego 49, 60-355 Poznan, Poland.

13–17 December. New York Hilton Hotel, New York. *40th Postgraduate Assembly in Anesthesiology.*
Information: Mr Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 30 East 42nd Street, Suite 1501, New York 10017, USA.

1988

11–13 May. Sydney, Australia. *5th International Dental Congress on Modern Pain Control.*
Information: Australian Convention and Travel Services (ACTS), G.P.O. Box 1929, Canberra, A.C.T. 2601, Australia.

22–28 May. Washington, DC. *9th World Congress of Anaesthesiology.*
Information: American Society of Anaesthesiologists, 515 Busse Highway, Park Ridge, IL 60068, USA.

15–16 September. Southampton. *Annual Scientific Meeting, Association of Anaesthetists of Great Britain and Ireland.*
Information: Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA, UK.

1 October. Pavia. *National Congress Societa Italiana di Anest, Rianimazione e Terapia Intensiva*
Information: Dr G. Conti, SIARRTI, Universita degli Studi, La Sapienza di Roma, Viale del Policlinico, 00161 Roma, Italy.

18–21 October. Perth *Annual General Meeting, Australian Society of Anaesthetists.*
Information: The Secretariat, ASA, P.O. Box 345, Paddington, New South Wales 2021, Australia.

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Association of Anaesthetists of Great Britain and Ireland

Annual Report of Council 1985–1986

1. Membership of the Association (as at 30.7.86)

The total membership of the Association is 5284 (5257).

Honorary	24	(26)
Ordinary	2112	(2186)
Junior	1720	(1719)
Corresponding	35	(35)
Overseas	994	(991)
Retired	321	(264)
Senior	19	(19)
Associate	14	(14)

(Last year's figures in brackets.)

There were 31 resignations during the year.

Deaths

Council records with regret the death of the following members:

Ball, H.C.J. (*Hants.*), Bourne, S.P. (*Essex*), Conway, C.M. (*London*), Curry, F.E. (*Dublin*), Dunkin, L.J. (*Newcastle-upon-Tyne*), Durrans, S.F. (*Dorset*), Fernando, J.C. (*Sri Lanka*), Frazer, R.A. (*Bucks.*), Gilmartin, T.J. (*Dublin*), Griffiths, H.R. (*Canada*), Haxholdt, B. (*Denmark*), Kelion, B. (*London*), Langton-Hewer, C.J. (*London*), Parke, F.W. (*Worcs.*), Phillips, M. (*Ealing*), Pinson, K.B. (*Australia*), Roper, R.E. (*Aughton*).

2. John Snow Silver Medal

Council recommended the award of the John Snow Silver Medal to: Professor Sir Gordon Robson, CBE.

3. Honorary Membership

Council recommended the following for election to Honorary Membership:

Dr O.P. Dinnick, Emeritus Consulting Anaesthetist, Middlesex Hospital; Dr D.D.C. Howat, Honorary Consulting Anaesthetist, St George's Hospital; Professor Sir Gordon Robson, CBE, Professor of Anaesthetics, Royal Postgraduate Medical School; Dr C.F. Scurr, CBE, MVO, Honorary Consulting Anaesthetist, Westminster Hospital.

4. Pask Certificate of Honour

Council awarded Pask Certificates to:

P. Cull, Esq, Director, Audiovisual Department, St Bartholomew's Hospital, London; Dr S. Lipton, OBE, Director, Pain Relief Foundation, Liverpool; Dr Ruth Mansfield, MBE, Honorary Consulting Anaesthetist, Brompton Hospital, London; Air Commodore A.J. Merrifield, QHP, RAF, Whittingham Professor of Aviation Medicine, Farnborough.

5. Annual Scientific Meeting of the Association, Leicester 12–13 September 1985

The Annual Scientific Meeting was held in the Leicester Polytechnic. A full scientific programme had been organised by the local committee of the Leicester Anaesthetists. There were 450 registrants. The John Snow Lecture was delivered by Dr D. Acheson, Chief Medical Officer, DHSS. Dr Acheson gave a review of the history of the outstanding contribution to public health of his predecessors in that office.

The social programme included a reception, given by the Lord Mayor of Leicester, in the De Montford Hall, and the Annual Dinner, which was held in the Grand Hotel.

6. Annual General Meeting 1985

The Annual General Meeting was held at the Leicester Polytechnic on Friday 13 September 1985. The President, Dr T.B. Boulton, was in the Chair. Over 150 members were present.

The proposal to award the John Snow Silver Medal to Professor T.J. Gilmartin was greeted with acclaim. The medal was presented to Professor Gilmartin in Dublin on a later occasion. Dr G. Jackson Rees was unanimously elected to Honorary Membership. Pask Certificates were awarded to: Dr W. Maurice Brown (*Belfast*); Dr Margaret L. Heath (*London*); Cyril Jones, Esq (*East Grinstead*).

The Minutes of the Meeting have been circulated to all members.

7. Linkman Conference 1985

The tenth Linkman Conference was held on 11 September 1985, with the President in the Chair; 160 Linkmen were present. Information regarding the move to and the appeal for 9 Bedford Square was given. The educational activities planned for the new building were explained, including the holding of seminars for up to 40 registrants, throughout the year. Other topics discussed during the day included the implementation of management proposals, private practice in National Health Service Hospitals, the Confidential Enquiry into Peri-operative Deaths, Recovery Facilities and Operating Department Assistants and their problems.

Professor A. Goldberg from the Committee on Safety of Medicines spoke on the reporting of adverse drug reactions by anaesthetists and a discusson followed. A discussion, based on the Association document 'Workload for Anaesthetists', and including many other topics, was held in the Open Forum. A detailed report of this Conference has been circulated to Linkmen, and published in *Anaesthesia*.

8. Postgraduate Study Day 1985

A successful Postgraduate Study Day was held jointly with the Faculty of Anaesthetists at the Royal College of Surgeons, on Saturday 19 October 1985. A similar format to that of previous years was followed. Eighteen lectures were delivered in three lecture theatres, allowing participants to hear six speakers of their choice. Some 130 people attended.

9. Future meetings

Association meetings

1986—Postgraduate Study Day, *18 October 1986*, Joint Meeting with the Faculty, Royal College of Surgeons of England, London.

1987—Symposium on Management for Anaesthetists, *17 January*, Royal College of Surgeons of England, London

—Junior Anaesthetists' Group Linkman Conference and Annual Scientific Meeting, *8–10 April*, Belfast.

—Association Linkman Conference, *9 September*, Sheffield.

—Annual Scientific Meeting and Exhibition, *10 and 11 September*, Sheffield.

1988—Junior Anaesthetists' Group Linkman Conference and Annual Scientific Meeting, *5–8 April*, Nottingham.

—Association Linkman Conference, *14 September*, Southampton.

—Annual Scientific Meeting and Exhibition, *15 and 16 September*, Southampton.

1989—Joint Meeting with the Canadian Anaesthetists' Society, *June*, Ottawa.

—Linkman Conference and Annual Scientific Meeting and Exhibition, *September*.

1990—Linkman Conference and Annual Scientific Meeting and Exhibition, Manchester

1993—Annual Scientific Meeting (Joint Meeting with the Canadian Anaesthetists' Society), *September*, Edinburgh.

Sponsored meetings

1987—The Second International Symposium on the History of Anaesthesia, *20–23 July*. The Symposium, which is sponsored by the Association, will be held at the Royal College of Surgeons of England, London. The preliminary programme and call for free papers has been widely circulated and considerable international interest has been generated. The Organising Committee look forward with confidence to a successful and interesting event.

Other meetings

1986—Royal College of Surgeons of England, Annual Meeting (Anaesthetic Section), *September 24–26*.

—The Asian Australasian Meeting, Hong Kong, *September*.

—Post Conference Meeting, Peking, *September*.

1988—9th World Congress of Anaesthesiology, *22–28 May*, Washington, DC, USA.

10. Council

During the year October 1985–September 1986, Council and its Advisory Committee have each met on five occasions. The attendances of members at Council and the Advisory Committee are shown later, together with the membership of the various subcommittees and working parties.

Council wishes to express its appreciation of the work done by Miss Ann Muir, Administrative Secretary, by Mrs Betty Tyler, her assistant, by Miss Pat Plant, the Financial Assistant and by our good friend Mr Ernest Warburton, who continues to give stalwart help to us in financial and legal matters.

Retiring from Council this year will be Professor M.D. Vickers, who has continued to contribute sage advice since his most successful Presidency. Professor J.P. Payne leaves after a term of two years as Vice-president, and before that several years as a co-opted member of Council, representing the Medical Academic Staff Committee of the B.M.A.

Three elected members of Council retire after serving four years: Dr R.J. Eltringham, Dr W.H.K. Haslett and Dr D.J. Hatch. All have contributed extensively to the work of the Association and have earned Council's gratitude.

Nine Bedford Square, London WC1

The Association Secretariat moved to 9 Bedford Square in November 1985 and quickly adapted itself to its new, larger and more comfortable environment. The year has seen the consolidation of the administrative machine and an improvement in efficiency with the aid of computerisation. The building is being furnished gradually and tastefully with the aid of funds raised by the Appeal. The house is already being used by many individuals and by medical organisations for scientific, social and committee meetings. The new series of Seminars promoted by the Association has begun and is proving very successsful. The official Opening Ceremony will take place in the Spring of 1987, by which time 9 Bedford Square will have become a well appointed and prestigious centre in London for the specialty of anaesthesia. The achievement of this objective represents the realisation of farsighted planning by a generation of Officers of the Association; it is for a new generation to exploit the potential to the full.

11. Linkman Organisation

The Linkman Organisation continues to be an important link between the Officers and Council and the Membership. A review of the list of Linkmen is being undertaken. Linkmen are asked to keep in touch with the office and notify any changes. Districts without a Linkman are asked to get in touch with the Hon. Secretary. Linkmen are particularly requested to share information sent to them by the Association with their colleagues and their Health District.

Members of the Association and Linkmen are welcome to write or telephone the Association Headquarters on any matter of concern or interest. A list of Linkmen is given at the end of this report.

12. Education and Research

The Committee (Chairman, Professor Graham Smith) met twice during the last year (1 November 1985 and 4 April 1986) and devoted much of its time to the distribution of grants authorised by Council.

Grants awarded 1985/86

Research grants. Dr. R.A.F. Linton (*St Thomas's*) £5000 for an investigation into the levels of plasma sodium during prostatectomy; Dr M. Coplans and Professor I. Curson (London) £250 for an enquiry into deaths associated with dentistry; Dr R.P. Keen (*Manchester*) £200 for the testing of cognitive functioning in operating room personnel following exposure to anaesthetic gases.

Travel grants

Dr R.S. Jones (*Birkenhead*) £200 to visit centres of Veterinary Anaesthesia in the USA; Dr K.C. Judkins £250 to visit burns centres in Australia, New Zealand and the USA; Dr D.McD. Coventry (*Glasgow*) £200 to visit centres practising regional anaesthesia in Scandinavia.

Important changes have taken place in the travel grants given by the Association. In 1986, it was decided that there should be two types of travel grant. Firstly, a travel grant awarded under the old regulations. This permits Members of the Association to visit centres for educational purposes, but does not provide funds for attending scientific meetings. In 1986 the value of this award was increased to £500. Secondly, a new award termed 'Award for educational visits to third world countries' was established. This will provide travelling support for anaesthetists who are spending extended periods of time working in third world countries on very low salaries. In the last year, four applications have been received for travel grants and three awarded. Three applications have been received for educational awards of which one was approved and further information is awaited on the other two, regarding applications for financial assistance from the Overseas Development Administration.

Following the resignation of Dr Fiona McLellan from the Association's Research Fellowship, the post was readvertised and five good applications were received. Outside assessors' reports were sought on three applications, following which two individuals were interviewed and the award was made to Dr Angela Cooper of Cambridge to undertake a study on 'Induced hypotension during neurosurgical operations'.

The Committee has been active in the organisation of various meetings. In association with the Representatives of the Faculty of Anaesthetists, the Programme for the Postgraduate Study Day was drawn up for 1986 and, for the present year, the arrangements will be administered by the Association. In addition, a programme of 'Management for anaesthetists' has been arranged for January 1987.

Seminars at 9 Bedford Square

Three seminars have been held at our new headquarters. Three very different topics were chosen to try a seminar environment limited to 30 people, to encourage discussion. The topics have been: The Conduct of Private Practice, The Management of Head Injuries, and Analgesia for the Neonate.

Lunch and tea were provided and, in view of the success of these seminars, a full programme for 1986–7 is being arranged.

Educational visits to third world countries

Dr Celia Allison £750 to take up an appointment at the Children's Hospital, Kathmandu, Nepal.

Undergraduate prize essays

1st Prize—£100 S.K. Hogan (*Leeds*); 2nd Prize—£50 G. Matfin (*Leeds*); 3rd Prize—£25 Deborah A. Wales (*Leeds*).

13. Safety Committee

Meetings of the Safety Committee (Chairman, Dr P.W. Thompson) were held on 19 July 1985, 13 December 1985 and 6 June 1986. The question of earth-free power supplies and double-pole fusing in operating theatres was finally resolved, in agreement with the Department of Health and Social Security (DHSS) and the British Anaesthetic and Respiratory Equipment Manufacturers' Association.

Other matters which have been discussed (often not for the first time) were breathing system disconnexions, the labelling of ampoules and problems of electrical safety in the presence of flammable gases. A wide variety of queries from members have been dealt with and, when appropriate, referred to the DHSS or other bodies. The Association has continued its deep involvement in the preparation of International and National Standards for Anaesthetic Equipment and progress has been very good during the past year.

14. Report of International Relations Committee

The International Relations Committee (Chairman, Dr R.J. Eltringham) met on two occasions. The existing programme of cooperation and aid for the developing countries was continued and, in response to requests, expanded considerably.

Dr A. Tomlinson (*Stoke on Trent*) conducted the annual two months training course in Nepal and arrangements have been completed for Dr Celia Allison (*Rotterdam*) to replace Dr Angela Barlow for a year's lectureship at Kathmandu Children's Hospital. Dr D. Harris (*St. Thomas's Hospital*) spent three months lecturing in the Sudan and Dr Magdi Mcharib visited the United Kingdom as part of the exchange programme between the Universities of Bristol and Khartoum.

Dr R. Sinclair (*The Brook Hospital*) has been so successful in resuscitating the Department of Anaesthesia in Lusaka that he has decided to stay on for a second year, during which he will be assisted by Dr I. Wilson (*Plymouth*).

The exchange programme with East Germany (DDR) continued with a visit to the United Kingdom by Dr Ingrid Hörning (*Cottbus*), and Dr R.J. Eltringham (*Gloucester*) visited the DDR. Further visits by members of both societies are being encouraged.

The requests for help in setting up training programmes at the Universities of Kumasi (Ghana) and Harare (Zimbabwe) have both been met following exploratory visits by Dr J.B. Broadfield (*King's*) and Dr Eltringham (*Gloucester*). Dr B. O'Donoghue (*Nottingham*) has begun a one year appointment in Kumasi and is shortly to be joined by Dr R. Page (*Plymouth*). Lecturers are currently being sought for Zimbabwe and three training posts have been arranged in the United Kingdom to enable Zimbabwean trainees to prepare for the FFARCS examination.

Dr Patricia Coyle reported from Uganda that conditions are settling down following the latest coup and she feels it is now safe to begin to expand the anaesthetic training programme in that troubled country.

The WFSA hopes to mount a refresher course in Nairobi, which, if successful, could become an annual event. On the first occasion it is planned to mount the course immediately before or after the annual meeting of the East African Society of Anaesthesiologists so that participants will be able to attend both events. Subsequent courses may be held in different parts of eastern or southern Africa, e.g. Zambia, Zimbabwe or Tanzania. Members of the Association will undoubtedly be called on to assist with this venture. Further requests for help have been received from Malawi, Tanzania, Nigeria and Kwazulu (S.Africa) and these will be explored during the coming year.

There has been so much interest in the course for anaesthesia in the developing countries organised annually by Dr M. Dobson and Dr G. Patterson in Oxford that a second course is being mounted in October by Dr Carter and Dr J.S.M. Zorab at Frenchay Hospital, Bristol.

15. Editorial Board

The Editorial Board (Chairman, Dr P.J. Helliwell) met on two occasions. Diligent work by all concerned in production of the journal has ensured that advance copies of *Anaesthesia* are available for distribution between ten and fourteen days prior to the cover date. It is now clear that the delays in the delivery of journals can only rest in the postal system. All other causes have been eliminated.

The journal continues to attract authors from throughout the world and the Board has noted the continuing steady improvement in the quality of material published. Pressure of work on the Assistant Editors has resulted in the decision to recommend that a fifth assistant editor be appointed. No changes in active personnel have occurred during the year, but the Board received the news of the death of the Founding and Emeritus Editor of *Anaesthesia*, Dr C. Langton Hewer, with deep regret.

16. Finance Committee

The Finance Committee (Chairman, Professor M. Rosen) has met twice in 1985–1986. A full review of the Association's finances, capital and current expenditure was undertaken to examine the effects of the major changes in process. The Association and The Education and Research Trust are now limited liability companies, the latter being a charitable company.

The Finance Committee considered that the Association's financial position was sound and at present capable of meeting future requirements. The success of the Appeal will ensure the completion of refurbishment and furnishing of 9 Bedford Square as well as the implementation of a new educational plan. The Finance Committee once more recorded its gratitude for the outstanding advice and services of Mr E. Warburton, Financial Adviser.

17. Private Practice Committee

The Private Practice Committee (Chairman, Dr M.T. Inman) recommended to Council the new guidelines for anaesthetic fees in private practice. These were published in January 1986 and have been distributed widely to Linkmen, members who ask for them and the Provident Associations. The separation of surgeons' and anaesthetists' benefits by the Provident Association and other health insurers has occasioned continued discussion and such provision is being actively sought by this Association.

A paper on the conduct of private practice by anaesthetists, covering topics such as group practice and continuity of care and private practice in National Health Service hospitals, is being prepared. A very successful seminar, touching on some of these subjects was held in May 1986 at 9 Bedford Square.

18. Junior Anaesthetists' Group

During the past year the Junior Anaesthetists' Group (Chairman, Dr G.W. Hamlin) of the Association has grown to 1720. The committee has been involved in two major projects in addition to routine committee work. Dr Wendy Scott has been concerned in the preparation of a new book designed to assist those beginning a career in anaesthesia by providing advice and information on the specialty. Dr N. Redfern has been the organiser of a questionnaire sent to all registrars in anaesthesia, covering many aspects of their employment.

The Junior Linkman scheme, started last year, has proved very successful under the control of the Vice-Chairman and Linkman Coordinator, Dr A.D.J. Nicholl. The second annual meeting was held in association with the Annual Scientific Meeting and was attended by 43 Junior Linkmen.

The Scientific meeting this year was at Cardiff on 8–10 April. An excellent scientific and social programme was organised by the local committee. It was unfortunate that the Registrars' Prize was poorly subscribed and the quality of entries was not as high as in previous years. It is hoped that the standard will improve next year.

At the end of the meeting, the Chairman for the last two years, Dr Wendy Scott, retired. Her successor is Dr G.W. Hamlin and Dr Lauren Allan has been elected Secretary.

Next year's Scientific meeting will be at Queen's University, Belfast, 8–10 April 1987.

19. Confidential Enquiry into Peri-operative Deaths

The joint study between the Association of Anaesthetists of Great Britain and Ireland and the Association of Surgeons of Great Britain and Ireland, the office of which is housed in 9 Bedford Square, has now started in three health authority regions in the United Kingdom (the Northern, the South Western and the North East Thames regions). More than 95% of consultant anaesthetists and consultant surgeons are co-operating and about 300 assessors are involved. Every hospital in the three Regions has corporately agreed to take part. The Nuffield Provincial Hospitals Trust and King Edward VII Hospital Fund for London have added supplementary grants to their already generous grants.

The working party has met once in the past year under the Chairmanship of Dr E.A. Cooper as Vice-Chairman in the absence of the Chairman of the Committee, Professor M.D. Vickers. It is anticipated that, on the basis of results achieved so far, this unique study will reveal information about mortality following about half a million operations and will be concerned with about seven or eight thousand deaths.

20. CCHMS Anaesthetic Sub-committee

This brief report covers two years, October 1984 to September 1986. The Sub-committee (Chairman, Dr M.T. Inman), which met on three occasions, has four representatives of the Association and four representatives (anaesthetists) of the CCHMS, together with a representative of the Faculty of Anaesthetists and the JAG and the HJSC.

The Committee has continued its interest in health districts which have had difficulties in staffing. Domiciliary visits by consultant anaesthetists who have had difficulties in obtaining payment have been discussed and resolved.

A cautious welcome was given to the 33% increase in NHS dental anaesthetic fees, but the committee remains concerned at the overall low level of these fees. Junior anaesthetists particularly remain concerned about the inadequacy of compensation for injury or illness attributable to work, provided by the NHS Injury Benefit Scheme, and have noted that some health authorities have taken out additional cover.

The Committee also discussed the question of direct payment of fees by Provident Associations and the problems caused by the under insurance of some patients. The Sub-committee welcomed the decision of the Joint JCC/CCHMS Sub-committee on Independent Medical Practice to investigate and publish guidelines for fees for all hospital specialties.

21. Working Parties

Specialist Anaesthetic Societies. A meeting with Specialist Societies in Anaesthesia and Intensive Care was held in November 1985. This meeting was attended by members of all such societies in the UK and general matters of communication and dates for meetings were discussed.

Smoking and Anaesthesia (Chairman, Professor M. Rosen). A small working party to examine the effects of smoking and anaesthesia met with representatives of the Health Education Council. It was decided to produce two papers to bring forward to the Linkman Conference. One, a paper on the subject for health professionals, to stress the value of health education in this field and the other for the patient who wishes to know more about the subject. These papers, if approved, will be sponsored by the Health Education Council.

Working Party on Smaller Obstetric Units (Chairman, Professor M. Rosen). Council has set up a joint Working Party with the Obstetric Anaesthetists' Association, to consider anaesthetic services for small obstetric units. The Working Party has met on two occasions and guidelines are being prepared outlining acceptable levels of anaesthetic service for small obstetric units. This document should be available in early 1987.

Your Anaesthetic. Our publication 'Your Anaesthetic' has been revised and reprinted and is available from the Association offices. It is designed to inform and reassure patients about to undergo general anaesthesia.

22. The Resuscitation Council UK

The Resuscitation Council (Representative, Dr P.J.F. Baskett) is composed of individuals from all specialties with a legitimate interest in cardiopulmonary resuscitation (CPR). It has produced published Guidelines in Basic Life Support (Posters and Stickers), thanks to generous grants and sponsorship from the British Heart Foundation and philanthropic companies. A videotape on Advanced Life Support is planned to become available in early 1987.

Representatives of Council attended the meeting of the American Heart Association, in Dallas, in mid-1985, to review and revise their resuscitation standards and contributed substantially to the debate. The revised American Standards are almost identical to the Resuscitation Council's Guidelines.

The Council has been closely associated with the BBC's 'Save-a-Life' Campaign—a series of short television broadcasts to be shown in the autumn of 1986, which depict the action to be taken by members of the public in a variety of emergency situations. The broadcasts will be supported by a coordinated, nationwide opportunity for the public to undertake short courses in CPR. These courses will be provided by the voluntary aid societies, various charitable bodies, the ambulance service and other competent supportive groups. Further details of this project and other activities of the Resuscitation Council are available from the Hon. Secretary, Dr D. Zideman, Department of Anaesthesia, The Hammersmith Hospital, Ducane Road, London.

23. The Monospecialist Committee for Anaesthesia and Reanimation for the EEC

The Monospecialist Committees (Association Representative, Dr P.J.F. Baskett) consist of two representatives from each EEC member country and seek to maintain and improve the standard of specialty training and practice throughout the community. Recommendations have been made regarding the period of postgraduate training, the role of the anaesthetic nurse and guidelines for the facilities and equipment required for safe anaesthetic practice. Discussion continues concerning EEC recognition of the existing specialist diplomas, such as the English and Irish Fellowships and the Diploma of the European Academy of Anaesthesiology.

Two meetings of the Committee have been held during the past year—in Lille in the autumn of 1985 and in Copenhagen in the spring of 1986. Dr D.D.C.

Howat has now retired from the Presidency of the Committee, after many fruitful and constructive years in the Chair. He will be succeeded as President by Dr P.J.F. Baskett, now the member nominated by the Board of Faculty of Anaesthetists. Dr W.R. MacRae has been chosen as the other UK representative nominated by the Association of Anaesthetists.

Composition of Council, its Sub-Committees and Working Parties

Council

Five meetings held (attendance in brackets).

Officers: Dr T.B. Boulton, President (5); Professor M.D. Vickers, Immediate Past President (2); Professor M. Rosen, President-elect (4); Professor J.P. Payne, Vice-President (3); Dr W.R. MacRae, Vice-President (5); Dr Jean Horton, Vice-President (working overseas);. Dr M.M. Burrows, Honorary Treasurer (4); Dr M.T. Inman, Honorary Secretary (4); Dr P. Morris, Assistant Honorary Secretary (4); Dr J.N. Lunn, Editor (4).

Elected Members: Professor A.P. Adams (3); Dr K. Budd (3); Dr Elizabeth Bradshaw (4); Dr R. Eltringham (5); Dr W.H.K. Haslett (4); Dr W.L.M. Baird (5); Dr D.J. Hatch (4); Professor W.S. Nimmo (4); Dr T.W. Ogg (4); Dr J.F. Searle (4); Professor G. Smith (3); Dr W.S. Wren (2); Dr W. Scott (4); Dr W. Hamlin (4); Dr L. Allan (1) (since April 1986).

Co-opted Members: Dr Aileen K. Adams; Dr P.J.F. Baskett; Professor C.M. Conway; Dr P. Keane; Dr P.J. Helliwell; Dr E.B. Lewis; Group Captain C.A.B. McLaren; Dr P.W. Thompson; Dr J.S.M. Zorab.

Advisory Committee

Five meetings held (attendance in brackets).

Chairman: Dr T.B. Boulton (5); Professor M.D. Vickers (overseas); Professor M. Rosen (5); Dr M.M. Burrows (5): Dr R.J. Eltringham (3); Dr W.R. MacRae (4); Dr M.T. Inman (5); Dr Aileen K. Adams (4); Dr J.N. Lunn (5); Dr P. Morris (5); Dr J.F. Searle (2); Professor G. Smith (4); Dr Wendy Scott (4).

Editorial Board

Chairman: Dr P.J. Helliwell; *Editor:* Dr J.N. Lunn; *Advisory Editors:* Dr R.S. Atkinson, Dr T.B. Boulton, Dr P.J.F. Baskett, Dr C.F. Scurr, Dr D.J. Hatch (Council Representative), Dr Lauren Allen (JAG); *Assistant Editors:* Dr C.E. Blogg, Dr R. Greenbaum, Dr R.M. Jones, Dr M. Morgan; *Co-opted Members:* Mr E. Warburton (Financial Adviser); Miss Joan Fujimoto (Academic Press); Dr Sue Silver (Academic Press); Mrs S.M. Campbell (Editorial Assistant).

Finance Committee

Professor M. Rosen (*Chairman*); Dr T.B. Boulton, Dr K. Budd. Dr M.M. Burrows, Dr P. Morris, Dr J.F. Searle, Dr J.N. Lunn, Mr E.Warburton.

Safety Committee

Dr P.W. Thompson (*Chairman*); Professor A.E. Adams, Dr Elizabeth Bradshaw, Dr D.J. Hatch, Dr T.W. Ogg, Dr A.D.J. Nicholl (JAG), Dr J. Bushman, Dr Jean Lumley, Air Commodore A.J. Merrifield, Professor M.K. Sykes, Dr Kate Allsopp (MDU), Dr [illegible]. Hickey (MPS)

Co-opted: Mr L.W.M. Arrowsmith (DHSS), Mr C. Bray (DHSS), Mr M. Freeman (BAREMA).

Education and Research Committee

Professor G. Smith (*Chairman*); Dr Jean Lumley, Professor W.S. Nimmo, Dr P. Morris, Dr T.W. Ogg, Professor J.P. Payne, Dr Wendy Scott (JAG until April 1986), Dr G.W. Hamlin (JAG from April 1986).

Junior Anaesthetists' Group

Dr Wendy Scott (*Chairman* until April 1986); Dr G.W. Hamlin (Secretary until April 1986, Chairman thereafter), Dr Lauren Allan (Secretary from April 1986), Dr A.D.J. Nicholl (Vice-Chairman from April 1986), Dr Anne Blyth, Dr L.E. Conroy, Dr Nancy Redfern, Dr Deborah Schiller, Dr I Woods, Dr K.G.Lowry, Dr G.J. Fitzpatrick, Dr S.P. Linter, Dr D. Paul. Representative of Council: Dr R.J. Eltringham.

Central Committee for Hospital Medical Services

Dr M.T. Inman.

Anaesthetists' Sub-committee of the CCHMS

Dr M.T. Inman (*Chairman*); Dr T.B. Boulton, Dr P. Morris, Dr W.H.K. Haslett, Dr Deborah Schiller (JAG).

International Relations Committee

Dr R.J. Eltringham (*Chairman*); Dr Aileen K. Adams, Professor A.P. Adams, Dr Elizabeth Bradshaw, Dr D.J. Hatch, Dr W.S. Wren, Dr J.S.M. Zorab, Dr W.L.M. Baird, Dr M. Dobson, Dr D.J. Hatch, Dr Wendy Scott, Dr I. Woods. *By invitation, representatives from:*

Appropriate Health Resources and Technologies Action Group, British Council, Inter-University Council, Bureau for Overseas Medical Services, Overseas Development Administration, World Federation of Societies of Anaesthesiologists, Committee for International Co-operation in Higher Education.

Confidential Enquiry into Peri-operative Deaths

Professor M.D. Vickers (*Chairman*); Dr M.M. Burrows, Mr J.E. Craven, Mr H.B. Devlin, Mr D. Harper, Dr M.T. Inman, Professor J. Lumley, Dr J.N. Lunn, Professor A.G. Johnson, Dr P. Morris, Dr D.B. Scott.

Private Practice Committee

Dr M.T. Inman (*Chairman*); Dr P.J. Baskett, Dr K. Budd, Dr R.J. Eltringham, Dr T.W. Ogg.

Organising Committee of the 2nd Symposium on the History of Anaesthesia

Dr T.B. Boulton (*Chairman*); Dr P.J.F. Baskett (*Secretary General*), Dr M. Rosen, (*Honorary Treasurer*), Dr R.S. Atkinson (*Deputy Chairman*), Dr R.H. Ellis, Dr I. McLellan, Dr G.M.C. Paterson, Dr P.W. Thompson, Dr N. Newton.

Working Party on Anaesthetic Services for Small Obstetric Units

Professor M. Rosen (*Chairman*); Dr W.L. Baird, Dr Elizabeth Bradshaw, Dr Barbara Morgan, Dr Rosemary MacDonald, Dr Nancy Redfern, Dr Wendy Scott, Group Captain C.A.B. McLaren, Dr J. Thorburn, Dr P. Morris.

Working Party Between the National Representative Bodies in Anaesthesia and the Specialist Organisations

Dr T.B. Boulton (*Chairman*); Prof. A.P. Adams, Dr D.J. Hatch, Dr A.K. Adams, Dr G. Bush, Dr W. Fitch, Dr Constance Howie, Dr P. Morris, Dr Y.M.A. Tai, Dr G.J. Taylor, Dr T. Thomas, Dr R.D. Latimer, Dr Sheila Willatts.

The Officers of the Association are Ex-Officio Members of all Committees and Working Parties.

Faculty Advisory Panel on the Training of Anaesthetists

Dr G.W. Hamlin (JAG).

Liaison with BSI

Dr P.W. Thompson.

Joint Committee on the Higher Training of Anaesthetists

Dr W.R. MacRae, Dr G.W. Hamlin (JAG).

EEC Mono-Specialist Sub-committee of the UEMS

Dr P.J.F. Baskett.

National Resuscitation Standards

Dr P.J.F. Baskett.

Manpower Advisory Panel (DHSS)

Professor G. Smith, Dr G.W. Hamlin (JAG).

Association Linkmen

Names are grouped according to Regional Health Authority (Area in Scotland); the order of names within each group is arbitrary and without significance.

England

Northern (R.H.A. 1)

D.J.H. Daniel (*Hartlepool*); I.F. Riddle (*South Teeside*); F.B. Buckley (*Carlisle*); R. Goodwin (*Durham*); D.C. Townsend (*South West Durham*); K.S. Cameron (*North West Durham*); C. Beeton (*Darlington*); R.I. Anderson (*Hexham*); D.J. Greaves (*Ashington*); M.R. Bryson (*Newcastle Central Section*); S.K. Greenwell (*North Tyneside*); B.B. Bhala (*South Tyneside*); A.E. Arrowsmith (*Sunderland*); M. Marshall (*Newcastle Western Section*); P. Copeland (*Newcastle Eastern Section*); G. Harris (*North Tees*); K.C. Misra (*South Cumbria*).

Yorkshire (R.H.A. 2)

T.D. Biswas (*Pontefract*); S. Firn (*Wakefield*); J.M. Roberts (*Leeds University Hospital*); T.M. Jack (*Leeds General Infirmary*); J.P. Payne (*Dewsbury*); L.A.M. Sharaf (*Huddersfield*); F.W. Oliver (*Halifax*); S.G. Jones (*Airedale*); M.J. Wade (*Bradford*); G.J. MacWhirter (*Harrogate*); J.D. Kinnell (*York*); G.W. Whitfield (*Scarborough*); P.D. Winder (*Hull*); J.M. Clark (*Grimsby*).

Trent (R.H.A. 3)

A. Padfield (*Hallamshire Hospital, Sheffield*); G.P.M. Clark (*Northern General Hospital, Sheffield*); P.J. Mawson (*Rotherham*); B. Milne (*Doncaster*); P.S. Gadgil (*Barnsley*); C.D.D. Hutter (*Nottingham*); A.J. Whitaker (*Mansfield*); J.S.W. Thompson (*Scunthorpe*); J.R. Bowers (*Lincoln*); I. McLelland (*Leicester*); P. Cartwright (*Derby*); M.J. Wolfe (*Chesterfield*); W.S. Nimmo (*Worksop*); M. Bexton (*Boston*).

East Anglia (R.H.A. 4)

A.J. Pearce (*Newmarket*); A.E. Gray (*Norwich*); S.G. Harrison (*Ipswich*); J. Jenkins (*Great Yarmouth*); R. Francis (*King's Lynn*); A.F.M. Little (*Peterborough*); J.M. Collis (*Papworth*); C.W. Glazebrook (*Cambridge*).

North West Thames (R.H.A. 5)

P.J. Bennett (*Middlesex*); S. Saville (*Kensington and Chelsea*); P.F. Knight (*St Mary's*); B. Sanderson (*St Stephen's*); R.L. Hargrove (*Westminster*); R.E. MacLaurin (*Harefield*); W.J. Hegarty (*Hillingdon*); H.L. Clarke (West Middlesex); M. Meurer-Laban (*King Edward VII, Ealing*); J.G. Whitwam (*Hammersmith*); J. Stevens (*Charing Cross*); D.C. White (*Northwick Park*); I.F. Collier (*Central Middlesex*); P.J. Watters (*Edgeware*); A.E. Nicol (*Barnet*); I. McCallum (*Stevenage*); B. Pirie (*Welwyn*); R.W. Gabriel (*Luton*); R. Chakrapani (*Bedford*); A.R. Deacock (*Royal National Orthopaedic*); G.M.D. Archer (*Royal Marsden Hospital*); J. Ormrod (*University College Hospital*); D. Ellis (*St Peter's and St Paul's*); E.F. Battersby (*Great Ormond Street*); B.M. Dempsey (*Ashford*); H. Turner (*Watford*).

North East Thames (R.H.A. 6)

S.K. Coombes (*St Andrew's*); D. Wilkinson (*St Bartholomew's Hospital*); C.J. Stephens (*Epping*); D.G. Rees (*Whipps Cross*); M.G. Rolfe (*The London Hospital*); N.H.A. Doctor (*Hackney Hospital*); S. Helwa (*Enfield*); P.H. Simmons (*Royal Northern and North Middlesex*); M.S. Pegg (*Royal Free*); M. Bashir (*Barking*); G.B. Rushman (*Southend*); P.O. Bodley (*Romford*); R.W. Griffin (*Colchester*); R.A. Spilsbury (*Chelmsford*); D.A. Thomas (*Harold Wood Hospital*); L. Chippendale (*Whittington Hospital*); S.D. Dalal (*District Hospital*).

South East Thames (R.H.A. 7)

A.R. Thompson (*Hastings*); P.H. Venn (*Eastbourne*); T. Murray (*Brighton*); A. Proctor (*Canterbury*); E.B. Lewis (*Ashford*); H. Singh (*Dartford*); R.V. Sturton (*Bromley*); P.E. Daly (*Greenwich*); M.A. Thompson (*Lewisham and North Southwark, Guy's Hospital*); I.M. Corall (*King's College*); J.A. Mathias (*St. Thomas's*); C. Barham (*Kent and Sussex*); F.E. Bennetts (*Medway*); P.B. Wemyss-Gorman (*Cuckfield*).

South West Thames (R.H.A. 8)

B.M. Dempsey (*Ashford*); R.C. Brookes (*Croydon*); S.S. Barker (*Guildford*); D.A. Wright (*Kingston*); R. Waters (*Worthing*); J. Missen (*Chichester*); J. Lees (*Sutton and West Merton*); D.C. Hughes (*Surrey*); E.P. Berwick (*St George's*); A.M. Rollin (*Epsom*); I. Sutherland (*Frimley Park*).

Wessex (R.H.A. 9)

K.J. Gill (*West Dorset*); R.N. Packham (*Bournemouth and Poole*); C.D.G. Evans-Prosser (*Basingstoke*); R.W.D. Clunie (*Winchester*); A. Logan (*Isle of Wight*); P.W.R. Smethurst (*Salisbury*); R. Porteous (*Swindon*); D. Protheroe (*Bath*); A. Turrall (*Portsmouth*); J. Winder (*Southampton*).

Oxford (R.H.A. 10)

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South Western (R.H.A. 11)

J.A.I.T. Clement (*Bristol and Western Health Authority*); R. Weller (*Frenchay Hospital, Bristol*); J.T. Mulvein (*Southmead Health District, Bristol*); G. Jephcott (*Weston-super-Mare*); C. Harvey (*Cornwall and I.O.S. Health Authority*); P. W. Brighten (*Barnstaple*); W.B. Clarkson (*Exeter*); J. Thorn (*Torquay*); R.H.M. Hodgson (*Plymouth*); A.L. Burlingham (*Cheltenham*); T.A. Boliston (*Taunton*); F.G. Hall (*Yeovil*).

West Midlands (R.H.A. 12)

M.D. Milne (*Wolverhampton*); A. Leslie (*Walsall*); A. Patel (*West Bromwich*); V. Gnanadurai (*Stourbridge*); M.A.H. Lewis (*Coventry*); E.R.S. Hooper (*Good Hope*); P. Simpson (*East Birmingham*); J. Hurdley (*Sellyoak*); B. Barsham (*Dudley Road*); P. Jackson (*Queen Elizabeth*); D.G. Tweedie (*Warwick*); D. Eyre-Walker (*Stafford*); W.N. Ramsden (*Stoke*); C.T. Major (*Shrewsbury*); B. Head (*Bromsgrove*); I. Hine (*Hereford*); N.S. Kadusker (*Nuneaton*); N.O. Jones (*Worcester*); J.W. Martin (*Burton on Trent*).

Mersey (R.H.A. 13)

A.K. Ray (*North Wirral*); R.G. Snow (*Chester*); R.P. Howard (*Whiston and St Helen's*); J.C. Richardson (*Broadgreen*); T.H.L. Bryson (*Wirral*); E. Duane (*Warrington*); R.F. Graham (*Macclesfield*); F.M. Emery (*Leighton Hospital, Crewe*); W.F. White (*South Wirral*); C. Charway (*Southport*); Clare Howard (*Walton Hospital, Liverpool*); M.G.D. Salem (*Halton General Hospital*).

North Western (R.H.A. 14)

I.W. Jones (*Wigan*); S.V. Lees (*Trafford Health Authority*); J.H. Wright (*Ashton*); C. Pemberton (*Stockport*); C. Spanswick (*Salford*); P. Burridge (*Rochdale*); P. Davis (*Oldham*); H. Goldwater (*Crumpsall*); W.A. Thomas (*Withenshawe*); J.H. Breed (*Withington*); B. Foster (*Manchester*); M. Leahy (*Bury*); N.H. Naqvi (*Bolton*); W.A. Noble (*Preston*); K.M. Natrajan (*Blackpool*); M.A. Rucklidge (*Lancaster*); S. Mehta (*Burnley*); J. Fenwick (*Blackburn*); J.P.D. Friend (*Ormskirk*).

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Scotland

Highland

J. Muir (*Inverness*).

Western Isles

W.M. Speirs (*Stornoway*).

Grampian

R. Davidson-Lamb (*Aberdeen*); G.M. Duthie (*Elgin*).

Orkney

D. Johnstone (*Orkney*).

Tayside

P.B. Taylor (*Dundee*); A.A. Kutarski (*Perth*).

Fife

C.K. Adam (*East Fife*); J. Duncan (*West Fife*).

Borders

S. Robertson (*Peel Hospital*).

Lothian

L.V.H. Martin (*Edinburgh Royal Infirmary*); J.L. Jenkinson (*Western General Hospital*); J.C. McIntyre (*City Hospital and Southern Group*); N.A. Smith (*Princess Margaret Rose Orthopaedic Hospital*); A.E. Grace (*Bangour General Hospital*).

Glasgow

B.H. Maule (*Glasgow Royal Infirmary*); D.W. Proctor (*Glasgow Western Infirmary*); N.W. Lees (*Victoria Infirmary*); A. McKee (*Stobhill*); A. Mellon (*Glasgow Royal Infirmary*).

Lanarkshire

T.J. Nunn (*South Lanarkshire*); J.M. Thorp (*Airdrie*); G.A. Weetch (*East Kilbride*).

Forth Valley

D.H. Robertson (*Stirling*); W.J. Thomson (*Falkirk*).

Ayrshire

R.K.B. Young (*Kilmarnock*).

Dumfries and Galloway

J. Stubbs (*Dumfries*).

Argyll and Clyde

S. McKechnie (*Greenock*); T. Ireland (*Paisley*).

Northern Ireland

C. Ardill (*Belfast—The Ulster*); J.E. Hegarty (*Belfast—The City*); D.B. Wilson (*Belfast—R.V.H.*); W. McCaughey (*Craigavon*); I.H.C. Black (*Ballymena*); A.P. Jain (*Londonderry*.

Eire

R. Concannon (*Co. Mayo*); O. Comer (*Co. Galway*); D. Hogan (*Dublin*); W.S. Wren (*Dublin*); M. Lehane (*Co. Cork*); P. Fitzgerald (*Co. Limerick*).

Forces

Group Captain C.A.B. McLaren (*Swindon*).

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Safety Information Bulletin

(These are issued regularly by the Department of Health and Social Security from whom further information may be sought. The addresses are listed in Anaesthesia 1986; 41: 896).

Intravenous administration sets: disconnexion of components and bursting of delivery lines. SIB(86)65

Drip rate infusion pumps may generate excessive positive pressures within the delivery line of fluid administration sets. This may result in separation of components from the delivery line or may cause the tubing to swell and finally to split. For example, the tubing clamp should be placed between the drip chamber and the pump and not more distally. The increase in pressure within the tubing (as a result of distal obstruction) may occur slowly if the drip rate is set at a low level and the effects on the components or drip tubing may therefore be delayed for some considerable time.

Syringe pumps: uncontrolled infusion. SIB(86)63

Uncontrolled infusion or free flow from the syringe pump may occur if either the plunger or the barrel of the syringe is not attached securely to the appropriate part of the pump and the pump is placed at a significant height above the infusion site.

Unauthorised modification and adjustment of medical equipment. SIB(86)61

A thermocirculator had been modified to an extent that the temperature controller failed and also exposed staff to the risk of accidental electrocution. Two patients received severe burns and one patient subsequently died.

The Department again cautions against local, unauthorised modifications to equipment.

Thermega heating pads: risk of fire. SIB(86)58

There are dangers associated with these heating pads which result from a combination of poor design and inadequate servicing. Annual servicing is important.

Hazardous interconnexion of equipment due to incorrect usage of connectors or to human error. SIB(86)50

The Department continues to receive reports of hazardous interconnexions between various items of equipment, sometimes due to the use of inappropriate connectors. The Wright respiration monitor, Medical and Industrial Equipment (M&IE) monitoring equipment, Rigel/BOC monitors and some ECG patient leads have all featured in these reports.

Anaesthetic vaporizers: detachable types. SIB(86)45

Detachable anaesthetic vaporizers must be handled with extreme care. Where there is suspicion of damage to a vaporizer it should be returned to the manufacturer for checking and recalibration if necessary.

Silicone spiral reinforced hose used with breathing systems. SIB(86)47

The reinforced hose, used in patient breathing systems, may become delaminated and cause partial or total occlusion of the inner core. The integrity of the hose should be checked regularly.

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Erratum

Anaesthesia, 1986, Volume 41, pages 915–918

Postoperative nitrous oxide analgesia

Administration from air entrainment oxygen masks with a primary flow of Entonox

J. M. GODDARD

The symbols in the legend to Fig. 2 were reversed and should have been as follows:
I, Calculated using oxygen concentrations; ¦, calculated using nitrous oxide concentrations.

Notice to contributors to *Anaesthesia*

Manuscripts will be reviewed for possible publication on the understanding that they are being submitted to one journal at a time and have not been published, simultaneously submitted, or already accepted for publication elsewhere. This does not preclude consideration of a manuscript that has been rejected by another journal or of a complete report that follows publication of preliminary findings elsewhere, usually in the form of an abstract. Investigations performed on man must conform to appropriate ethical standards including voluntary, informed consent and acceptance by an ethical committee. Articles accepted become copyright of *Anaesthesia*.

Contributors are requested to submit two copies of manuscripts. They are also advised to retain a third copy as the Editors cannot accept responsibility for the loss of manuscripts in the post. The covering letter should be signed personally by all the authors and careful consideration should be given to the decision to include more than five authors. Articles should be forwarded to:

Dr J. N. Lunn, Editor of ***Anaesthesia****, Department of Anaesthetics, University Hospital of Wales, Heath Park, Cardiff CF4 4XW, UK.*

PREPARATION OF MANUSCRIPTS

Articles for *Anaesthesia* should be prepared in accordance with *Uniform requirements for manuscripts submitted to biomedical journals* (*British Medical Journal* 1979; **1:** 532–5) except that the titles of journals in the reference section should be given in full (see below). A reprint of these requirements of which this notice is a summary, can be obtained from the *British Medical Journal* price 50 pence (UK).

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The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36:** 119–25.

Anonymous. Epidemiology for primary health care. *International Journal of Epidemiology* 1976; **5:** 224–5.

BOOKS AND OTHER MONOGRAPHS

Personal author(s)

Osler AG. *Complement: mechanisms and functions.* New York: Prentice-Hall, 1976.

Corporate authors

American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. New York: Publishing Sciences Group, 1977.

Editor, compiler, chairman as author

Rhodes AJ, Van Rooyen CE, *comps. Textbook of virology: for students and practitioners of medicine and other health sciences*, 5th ed. Baltimore: The Williams & Wilkins Co., 1968.

Chapter in book

Weinstein L, Swartz MN. Pathogenic properties of invading micro-organisms. In: Sodeman WA Jr, Sodeman WA, eds. *Pathologic physiology: mechanisms of disease.* Philadelphia: W.B. Saunders, 1974: 457–72.

Agency publication

National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968—June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series* **10:** Data from the National Health Survey, No. 69) [DHEW publication No. (HSM) 72–1036].

OTHER ARTICLES

Newspaper article
Shaffer RA. Advances in chemistry are starting to unlock mysteries of the brain: discoveries could help to cure alcoholism and insomnia, explain mental illness. How the messengers work. *Wall Street Journal* 1977 Aug 12: 1(col 1), 10(col 1).

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Roueche B. Annals of medicine: the Santa Claus culture. *The New Yorker* 1971 Sept 4: 66–81.

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Journal of the Association of Anaesthetists of Great Britain & Ireland

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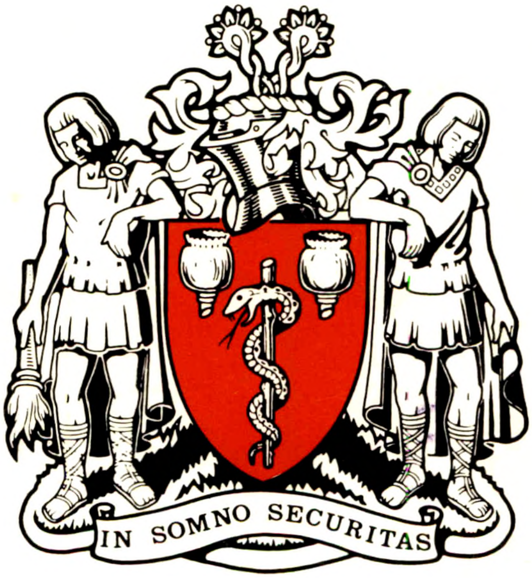

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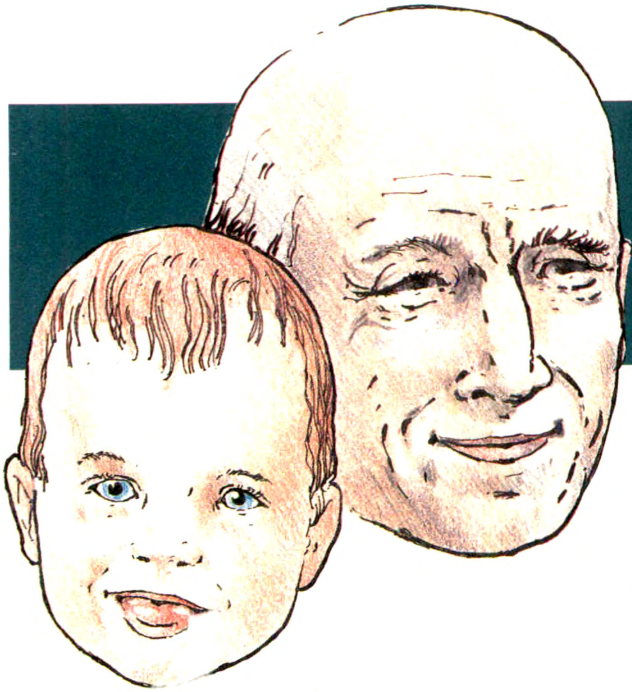

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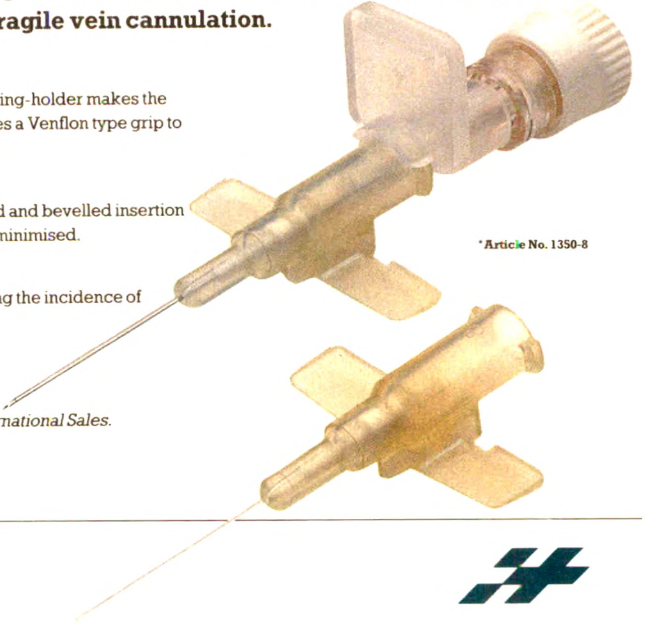

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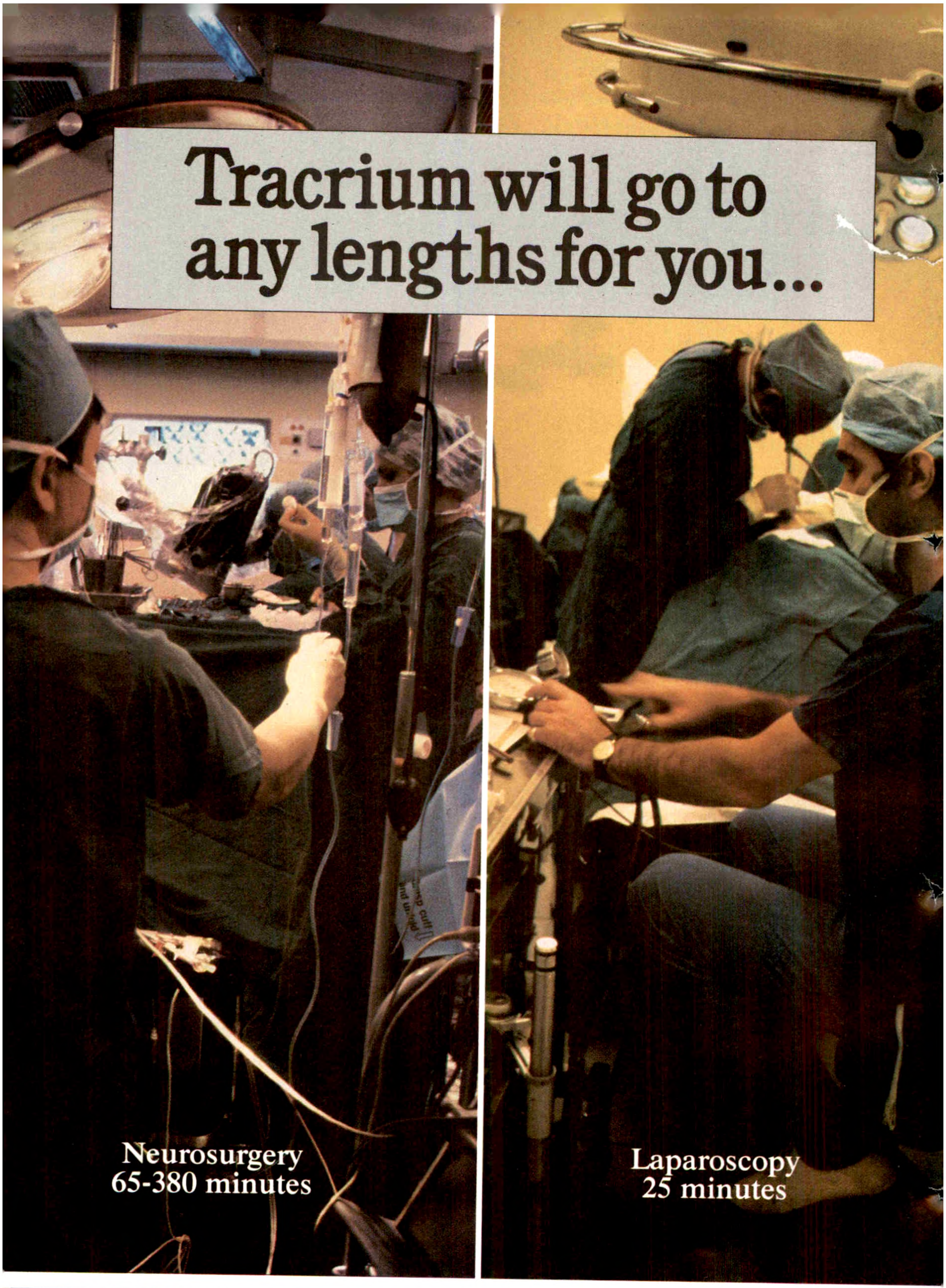
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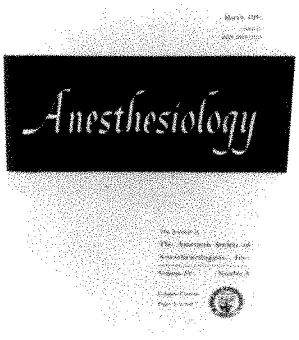

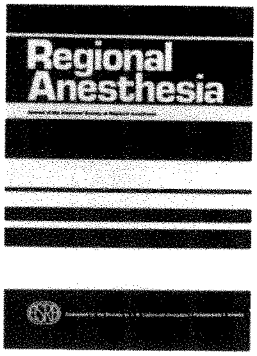

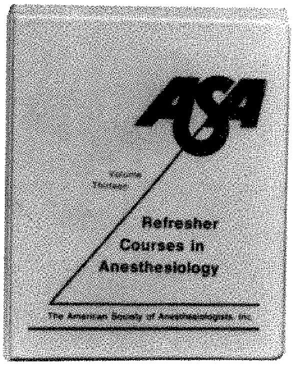

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Anaesthesia, 1987, Volume 42, pages 131–132

Editorial

The health of anaesthetists

Medicine is, arguably, the second oldest profession, but the specialty of anaesthesiology is a relatively young vocation. Perhaps this is the reason why the occupational health problems of anaesthetists have only recently received any degree of epidemiological scrutiny.[1]

The operating room carries obvious hazards for the patient, but the staff can also be at risk from fires and explosions due to the juxtaposition of flammable anaesthetic gases with electrical equipment. Resulting accidents are extremely rare but the gases themselves may have toxic properties of relevance to the anaesthetist. Halothane accumulates in the gonads and is a cause of hepatic hypersensitisation, methoxyflurane can damage sodium transport in the kidney whilst trichloroethylene is a possible carcinogen.

Despite all this, the evidence for an effect of such a working environment on the health of those employed in operating rooms is far from conclusive. Health statistics of UK anaesthetists for the period 1951–71 show an 8% deficit in all cancer mortality and an halving of the risk for lung cancer compared with the population as a whole.[2] A small excess of pancreatic tumours was, however, reported. A more recent mortality study of the records of the Faculty of Anaesthetists of the Royal College of Surgeons of England is underway and preliminary results were reported late in 1986. American data suggest that suicide may be in excess—a not uncommon finding in studies of medically qualified groups. Data from the UK (A. Neil, personal communication) are suggestive of a similar trend.

Perhaps the greatest interest has been shown in studies of pregnancy outcome in anaesthetists and their spouses. The literature here is extensive and conflicting. A recent review[3] concluded that many of the studies are flawed and subject to strong confounding and reporting bias, which severely limits the conclusions that can be drawn from the findings. There are some data to support the contention that there is an excess risk of spontaneous abortions among operating room staff and that the causative agent is nitrous oxide.[4] The evidence for an excess risk of congenital malformations is weak. No consistent excess for cancer exists in the extant studies whilst the studies of an occupational risk of hepatic and renal disease are conflicting.

Nevertheless, reports appear from time to time suggesting additional occupational risks from inhalational agents. Nitrous oxide, for example, is known to inactivate vitamin B_{12} by oxidation. A recent study of 21 dentists habitually exposed to nitrous oxide, suggested that deoxyribonucleic acid synthesis was suppressed in three, whilst two of these three had abnormal white cells in the peripheral blood films.[5] Similarly, a study of Norwegian anaesthesiologists reported a 'moderate incidence' of hepatitis B infections in anaesthesiology units—at least in part related to length and place of service.[6]

In most of these studies, gaseous atmospheric pollution has been invoked as a putative aetiological factor. In unscavenged operating rooms, nitrous oxide concentrations have been found to range from 108 to 430 p.p.m. with peaks as high as 5380 p.p.m. Halothane levels ranged from 1 to 8 p.p.m. with peaks of 115 p.p.m. Anaesthetists are not, of course, exposed to these levels for long periods of time but such levels can in the short term influence psychomotor performance and may have longer-term sequelae. The National Institute of Occupational Safety and Health (NIOSH) in the United States has recommended a standard that calls for the routine use of a series of scavenging and control measures to keep maximal concentrations of nitrous oxide below 25 p.p.m. in surgical operating rooms and below 50 p.p.m. in dental facilities. For halogenated anaesthetics the recommended limit is 2 p.p.m. alone and 0.5 p.p.m. in combination with nitrous oxide. Notwithstanding past exposure to high levels of anaesthetic gases, studies of anaesthetists do not appear to have shown incontrovertible evidence of serious health hazards. Thus the paper by McNamee and her colleagues in this issue (p. 133) is of particular interest—especially as it attempts to review morbidity patterns. A review of retirements among non-honorary consultant anaesthetists in English National Health Service hospitals suggested a two-fold excess rate of retirement on grounds of ill health, an excess of early retirements and an elevated number of deaths compared with surgical, medical, radiological and pathological specialty colleagues. No data are available on the causes for these morbid and mortal events—indeed, the authors had difficulty in collecting information on just two-thirds of the original study population. Nevertheless, these findings call for more detailed studies of morbidity in anaesthetists.

Epidemiological scrutiny of doctors by doctors may sound like self interest, but health care workers in general have received scant attention until recently. There is no doubt that the hospital environment harbours a number of suspect and proven hazards for those employed therein. Microbiological hazards are largely well described but less information is available on physical and chemical hazards. To date, the published studies on anaesthetists do not suggest that the hazards of the operating room are translated into serious health risks for the staff who work there. There are, however, some data to support the hypothesis that the health of anaesthetists may be affected in some degree by their job. The paper by McNamee and her co-workers adds to that concern but does not define the risks. Until further, more detailed, studies are undertaken, the occupational hazards of being an anaesthetist remain partly shrouded in the mists of the inhalational agents they administer.

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Editorial notices

Submission of manuscripts

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biomedical journals* (*British Medical Journal* 1979; **1:** 532–5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

News and Notices sections

The first issue of each quarter will contain full News and Notices sections.

Copy dates for these will be: 13 march for July 1987, 12 June for September 1987 and 11 September 1987 for January 1988.

Suppluementary News and Notices sections will be published in other issues but they will contain only material which requires publication before the next full News and Notices section.

Anaesthesia, 1987, Volume 42, pages 133–140

Morbidity and early retirement among anaesthetists and other specialists

R. McNAMEE, R. I. KEEN AND C. M. CORKILL

Summary

An historically prospective study of the rates of early retirement due to permanent ill health, early retirement between 60 and 64 years of age for other reasons, and deaths while in post, among consultant anaesthetists in England was carried out. The control group comprised consultants in four other hospital specialty groups. Approximately two-thirds of all consultants employed in the five specialties at National Health Service hospitals in England during 1966–83 were included in the study. Ill-health retirements among male anaesthetists were more than twice those expected on the basis of rates in the control group ($p < 0.001$). The number of other early retirements between 60 and 64 was a third higher than expected ($p < 0.005$). The number of deaths in post was also raised. For the smaller group of female anaesthetists there was not a statistically significant excess of ill-health retirements, but the number of early retirements between 60 and 64 was significantly greater than expected ($p < 0.005$), as was the number of deaths in post ($p < 0.001$). Possible causes of these excesses are discussed.

Key words

Manpower; anaesthetists.

In 1967 the results of a survey of morbidity among Russian anaesthetists were published.[1] There was no control group, but the rates for several conditions, including liver disorders, headaches, insomnia and spontaneous abortions among females, were thought to be high. It was observed that, besides exposure to anaesthetic gases, anaesthetists also had long and irregular hours of stressful work and were exposed to extremes of temperature and that some of these factors might be implicated.

Concern about the possibility of a health hazard in anaesthetic practice led to further studies and reviews.[2–7] Most of the epidemiological studies were primarily designed to investigate reproductive outcomes among anaesthetic staff and/or their spouses. Higher rates of spontaneous abortion among female theatre staff were frequently reported, but a recent review[7] concluded that the evidence of a causal relationship between anaesthetic gases and adverse reproductive outcome was inadequate, due to methodological problems in these studies.

Among other conditions which British anaesthetists reported more often than control groups were liver disease, lumbar disc disease, peptic ulcer and hypertension.[3] Again, methodological problems meant that these results were

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difficult to interpret. Information was usually obtained from questionnaires and often merely as an addendum to enquiries about obstetric history.[3] Respondents were aware of the aims of the study. Furthermore, questionnaire studies of working populations are an unsatisfactory method of investigating the incidence of serious diseases.[4]

The overall mortality experience of both British and American anaesthetists has been found to compare favourably with that of other doctors.[8,9] In a study in the United Kingdom,[8] the only excess noted was of deaths from pancreatic cancer, which the authors considered to be 'an artefact of chance thrown up in the course of examining more than 400 associations'. In the American study,[9] over 6% of deaths among male anaesthetists were attributed to suicide, compared with 1.5% of deaths among US male insurance policy holders.

Past evidence of an occupational hazard in anaesthesia, whilst inconclusive, suggested that further investigation was warranted. Furthermore, because of the problems of interpreting differences in rates of self-reported illness, more objective measures of morbidity were desirable. Employers' records of certified sickness absence were considered, but a preliminary examination indicated that they would be relatively incomplete for hospital doctors. It was therefore decided to compare the rates of early retirement due to permanent ill health among anaesthetists with those among other hospital doctors. Since consultants form one of the largest groups of hospital doctors, and are unlikely to change specialty after appointment, the investigation was limited to this group.

A pilot study[10] of consultants employed by two Regional Health Authorities indicated that basic information on those who had left posts, and their reasons for doing so, could be obtained from medical staffing and/or treasurer's records. However, information on the nature of ill-health retirements would not be made available on any account. Despite this drawback, it was considered that the investigation would be of value in providing a more reliable index of serious morbidity among anaesthetists than previously existed.

Materials and methods

All National Health Service (NHS), non-honorary, consultant anaesthetists who were employed by health authorities in England at any time during the period 1.1.1966–31.12.1983 were chosen as the study group. Honorary consultants were excluded because their commitments and work patterns were often very different from those of other consultants and because information on their retirements was not always available from health authorities. The control group comprised NHS non-honorary consultants belonging to surgical, medical, pathological, radiological, and radiotherapeutic specialties. Psychiatrists and other mental health specialists were excluded from the control group because the regulations governing their retirement were different from those for other consultants.[11]

In addition to the main comparison of the study, two others were made. Firstly, the rates of normal retirements at ages 60–64 among the study and control groups were compared. Under the NHS superannuation scheme, early retirement due to ill health entitles doctors to better pension benefits than those paid under normal circumstances.[11] However, these financial advantages do not apply to persons who have been members of the scheme for 20 years or longer, and are aged 60 or over. Such people would receive the same benefits regardless of whether retirement was normal or was granted on the grounds of ill health. It seemed likely, therefore, that some retirements where the actual reason was ill health would be included in the group of normal retirements at ages 60–64. Secondly, the numbers of deaths while still in post were compared.

All the health authorities in England were approached for permission to extract from their records, for all consultants in the study and control groups, the following information: date of birth, sex, specialty, date of appointment to post and, where relevant, date and reason for leaving a post (for example, death, new post, retirement due to ill health).

NHS re-organisations

During the period of study there were two re-organisations of the NHS, as a result of which coverage of the study and control populations was incomplete. The main problem was that, at the time of the re-organisations, the administrative units which held NHS consultant contracts changed. Records of consultants who had left those administrative units which were due to be abolished were in some cases passed on to new administrations, but in others they were lost or destroyed.

Consultants holding appointments with two or more authorities

Some consultants worked sessions for more than one employer, usually two or more authorities within the same region. For this analysis a consultant was considered to work only for that authority with whom the major commitment lay. When the commitments were equal, the allocation was made at random to one of the relevant authorities.

Analysis

Consultants were classified into ten subgroups, according to sex and specialty. Within each of these subgroups, the number of person-years at risk was calculated separately for each chronological period (e.g. 1966–70) by 5-year age groups (25–29, . . ., 60–64 years). As explained above, records of 'leavers' from some authorities were missing for part of the period of interest, usually the earlier years. In such cases, the remaining records for that authority were censored. For example, if the majority of records of consultants who had left an authority before 1970 were missing, then the number of person-years contributed before 1970 by consultants remaining in post after that date was set equal to zero.

For each sex, by age-group, by calendar period subgroup, the rates of early retirement due to ill health among consultant anaesthetists, and among all consultants in the control group, were calculated. For each sex separately, the rates among the study and control groups were compared using the methods described by Miettinen and Nurminen[12] for incidence–density data. This entailed the calculation of a Standardised Rate Ratio (SRR), which is a weighted average of the ratios of the rate in each study subgroup to that in the corresponding control subgroup, a Chi squared statistic for assessing the statistical significance of the difference between the SRR and unity and a 95% confidence interval (CI) for the SRR. Similar calculations were made for the comparisons with respect to normal, early (ages 60–64) retirements and deaths in post.

In a subsidiary part of the analysis, the rates of ill-health retirements, and other rates among the four specialties making up the control group were compared with each other. Here the observed number of retirements in each specialty was compared with the number expected on the basis of the overall rates in the control group; the comparison was summarised by a Chi squared statistic. The ratio of the observed to the expected number in each specialty is also a standardised rate ratio, although not strictly comparable to the SRR described above since different weights are used.

Results

Coverage

One Regional Health Authority and three teaching authorities refused to allow their records to be inspected. In addition, the records from one other region, five teaching authorities outside London and all but six of the teaching authorities in London, were either untraceable or were of such poor quality (one or more pieces of information missing in a majority of records) that they could not be used. Records from the remaining 12 regions and 14 teaching authorities mainly went back to 1966, but in some cases information on the earlier part of the period of interest was missing.

The extent of the coverage can be gauged by comparison with DHSS statistics on the number of consultants in post in England in September of each year. Table 1 shows the total number of permanent paid (i.e. non-honorary) consultants in the study and control populations for four of the years within the period of interest. Also shown are the corresponding numbers of consultants traced through our searches.

Comparison of anaesthetists with other consultants

The total person-years contributed by the male consultants included in the analysis was 93 482, of which anaesthetists contributed 13 220 (14%). Of the 116 early retirements due to ill health among male consultants, 26 were among anaesthetists. The standardised rate ratio (SRR) for this comparison was 2.15 ($p < 0.001$; 95% confidence interval, CI, 1.39–3.32). In Table 2, SRRs for ill-health retirements are given separ-

Table 1. Total numbers of permanent paid consultants in anaesthesia and control group and numbers traced.

	Year			
	1969	1973	1977	1981
Anaesthesia				
No. in post	1153*	1275*	1405	1556
No. traced	675	837	915	1078
% traced	59%	66%	65%	69%
Surgery, medicine, radiology and pathology				
No. in post	6324*	6802*	7479	8008
No. traced	3875	4571	5030	5645
% traced	61%	67%	67%	70%

Source: DHSS, Statistics and Research Division, Euston Tower.

* Only information on total numbers of consultants in post (permanent paid and honorary) was available for these years. The number of permanent paid consultants in each specialty was therefore *estimated* as the total number of consultants in each specialty times the percentage of consultants in that specialty in 1977 who were permanent paid staff.

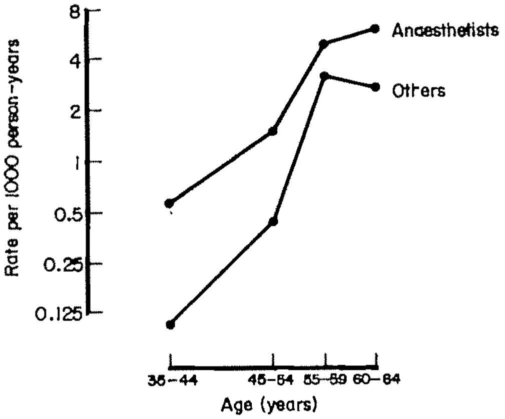

Fig. 1. Rates of ill-health retirements among male consultant anaesthetists and other male consultants by age.

ately for four age-groups and four time periods. The excess among anaesthetists was not confined to any particular age-group or time period, but it decreased with age up to the age of 60.

Among both anaesthetists and the other consultants, the rates of ill-health retirement increased exponentially with age (Fig. 1) up to the age of 60. After that age, the rate fell slightly among other consultants and increased only slightly among anaesthetists. This change in the rate of increase of retirements seems more likely to be due to changes in pension entitlement regulations at this age (see earlier), rather than to a change in the relationship between ill health and age. The number of normal early retirements between the ages of 60 and 64 among male anaesthetists was 105 and the SRR was 1.33 ($p < 0.01$; 95% CI, 1.09–1.64). As shown in Table 3, this excess was apparent throughout the period of interest, but appears to have been somewhat less in the later years.

There were 48 deaths among male anaesthetists while in post, with a corresponding SRR of 1.23 ($p > 0.10$; 95% CI, 0.91–1.68). The excess was confined to those under 60 years of age and to the period 1971–75 (Table 4).

The comparisons between female consultant anaesthetists and other female consultants were based on considerably fewer person-years than those for males. Total person-years for female

Table 2. Numbers of ill-health retirements among male anaesthetists and other male consultants, and Standardised Rate Ratios (SRRs) by age-group and time period.

	Age (years)				
	25–34	35–44	45–54	55–59	60–64
Anaesthetists	0	3	7	9	7
Others	0	3	13	45	29
SRR	—	5.22	3.29	1.47	2.19

	Time period			
	1966–70	1971–75	1976–80	1981–83
Anaesthetists	3	9	11	3
Others	11	23	38	18
SRR	1.95	3.00	2.32	1.07

Table 3. Numbers of normal early retirements at ages 60–64 among male anaesthetists and other male consultants, and Standardised Rate Ratios (SRRs).

	Time period			
	1966–70	1971–75	1976–80	1981–83
Anaesthetists	15	26	41	23
Others	87	151	281	202
SRR	1.48	1.66	1.28	1.11

Table 4. Numbers of deaths while in post among male anaesthetists and other male consultants, and Standardised Rate Ratios (SRRs) by age-group and time period.

	Age (years)				
	25–34	35–44	45–54	55–59	60–64
Anaesthetists	0	7	16	16	9
Others	2	30	81	78	90
SRR	—	1.26	1.26	1.59	0.91

	Time period			
	1966–70	1971–75	1976–80	1981–83
Anaesthetists	7	23	12	6
Others	65	86	88	42
SRR	0.83	1.96	0.95	0.99

consultants was 8107, of which 2600 (32%) were contributed by anaesthetists. The SRR of 1.22 ($p > 0.5$; 95% CI, 0.43–3.41) for ill-health retirements among female consultants was based on a total of only 15 retirements, five of which were among anaesthetists. The five anaesthetists were all under 60 years of age at the time of retirement.

The number of female consultant anaesthetists who retired normally between the ages of 60 and 64 was 31. The corresponding SRR was 1.75 ($p < 0.025$; 95% CI, 1.13–2.76). In both anaesthesia and the other specialties, the rate of retirement at these ages was much higher for females than males; the ratio of the rate for females to that for males was 1.7 for the non-anaesthetic specialties and 2.3 for anaesthetists.

There were eight deaths among female anaesthetists while in post and five among other female consultants. The SRR for this comparison was 3.96 ($p < 0.025$; 95% CI, 1.29–10.96). All eight anaesthetists died between the ages of 35 and 54 years.

Comparisons between other specialties

It was assumed in the preceding comparisons that, as far as rates of retirement and mortality were concerned, the four specialty groups (medicine, surgery, pathology and radiology) did not differ significantly from one another. However, it is possible that excessively high rates among one or more of the four groups might have been concealed because they were grouped together.

Table 5 shows the comparison between the observed (O) number of ill-health retirements in each of these four specialty groups and the corresponding expected (E) numbers for male consultants. There is no evidence of heterogeneity among the ratios of observed to expected numbers for these groups; in addition, these ratios for ill-health retirements and normal early retirements were very much less than the corresponding SRRs for anaesthetists.

Comparisons between the four smaller groups of female consultants were also carried out. The differences between the ratios of observed and expected numbers for ill-health retirements were almost statistically significant at the 5% level ($p = 0.052$); this was due to the excess number of ill-health retirements among pathologists ($O = 5$, $E = 1.71$, $O/E = 2.92$). There was no evidence of differences between the four specialties with respect to the ratios for normal early retirement or deaths while in post.

Table 5. Observed (O) and expected (E) numbers of ill-health retirements, normal early retirements (ages 60–64) and deaths in post among male consultants other than anaesthetists.

		Consultant group			
		Medicine	Surgery	Pathology	Radiology
Ill-health retirements	O	26	40	16	8
	E	29.54	39.85	10.98	9.36
	O/E	0.88	1.00	1.46	0.83
			χ^2(3df) = 3.00, p > 0.30 (SRR for anaesthetists = 2.16)		
Normal early retirements (60–64 years)	O	260	309	72	80
	E	254.67	317.17	78.40	70.76
	O/E	1.02	0.97	0.92	1.13
			χ^2(3df) = 2.05, p > 0.50 (SRR for anaesthetists = 1.34)		
Deaths in post	O	95	116	36	34
	E	93.27	127.36	31.63	28.73
	O/E	1.02	0.91	1.14	1.18
			χ^2(3df) = 2.62, p > 0.40 (SRR for anaesthetists = 1.24)		

Discussion

Male consultant anaesthetists have statistically significant excesses of both ill-health retirements and normal, early (age 60–64) retirements and they have a higher rate of deaths in post than the other specialties. The excess of ill-health retirements among female anaesthetists was not statistically significant, but the number of ill-health retirements among female consultants in general was small and so little can be concluded from this result. Female consultant anaesthetists did, however, have significantly increased rates of both normal early retirements and of deaths in post.

The possibility that these differences between anaesthetists and other specialists are a result of some inadvertent bias in the study methodology needs to be considered. The coverage of the study and control populations was incomplete (Table 1), but the main reason for this was administrative (NHS re-organisations) and so it is unlikely to be related to the outcomes under investigation. However, it could be argued that the other cause of incompleteness, nonparticipation by a few authorities, *was* related to these outcomes. However, if this is to explain the excess of retirements found in this study, then the anaesthetists employed by the nonparticipating authorities must have had much *lower* retirement rates than the other consultants. This seems unlikely.

It was thought that the calculated rates of retirement and mortality for consultants employed by London authorities might be less reliable than those for consultants employed elsewhere. Many London consultants had several concurrent employers and information was often not available from all of them. Accordingly, all analyses were repeated for London employees and non-London employees separately, but no differences were noted. In summary, therefore, it seems unlikely that incomplete coverage in this study has created a biased comparison between anaesthetists and other specialists.

No information on the nature of health problems or causes of death was available to this study, so other explanations can only be speculative. Hitherto, there have been two main explanations of apparent excesses in morbidity among anaesthetists: exposure to anaesthetic gases, and stress and fatigue. Evidence to support the former explanation is inconclusive; in particular, a dose–response relationship has never been demonstrated.[13] Although work in theatre is perceived to be stressful,[14] this area has received little attention apart from a rather limited investigation of biochemical responses in a small group of theatre staff.[15] The possibly related problem of drug abuse among anaesthetists has been investigated by Ward *et al.*[16] in America. They believed that chemical impairment was a particular risk of the specialty, but their survey had no control group. Thus, evidence to support the stress hypothesis is also tenuous.

If there is a causal relationship between anaesthetic gases and morbidity resulting in ill-health retirement, then three types of result might be expected in the present study. Firstly, assuming that the cumulative exposure of an anaesthetist tends to increase with age, the standardised ratios for ill-health retirements would increase with age. In fact, the standardised ratios for male anaesthetists *decreased* with age (Table 2). Secondly, given the growing use of scavenging systems in operating theatres over the study period, the standardised ratios should decline over time. However, Table 2 provides no evidence of this. Finally, because surgeons are also exposed to anaesthetic gases,[17] an excess of ill-health retirements should manifest itself in this group. Again, there is no evidence of this (Table 5).

As in other epidemiological studies of this type, the possibility that these results are due to selection rather than of environmental origin, needs to be borne in mind. Thus, it may be that doctors with a predisposition to poor health are attracted to anaesthesia with disproportionate frequency. Such a selection process would lead to an unhealthy worker effect, analogous to the better known healthy worker effect[18] found in some occupational groups. Furthermore, if, as has been suggested,[19] the effects of such selection processes on comparisons of morbidity diminish with time since selection, then the morbidity of anaesthetists should approach that of other doctors as their time in anaesthesia increases. The pattern of diminishing standardised ratios for ill-health retirements with age, as found in this study, is compatible with this proposition.

The present findings on mortality may reflect a genuine increase in the relative mortality of persons who had been employed as anaesthetists since the period (1951–71) covered by the last British mortality study.[8] However, the special nature of the present study, in which only working populations were compared, allows an alternative explanation. While we have found more deaths among male and female anaesthetists in post, it is possible that these were counterbalanced by greater longevity in those who survived beyond retirement. This suggestion is consistent with the age gradient found for the relative mortality of American anaesthetists,[9] that is, those under 55 years of age had a standardised mortality ratio (SMR) of 93, whereas it was only 80 for those who survived beyond this age. The chief cause of the higher relative mortality among American male anaesthetists under 55 years of age was suicide. The high rate of suicide among British medical practitioners in general (compared to the rest of the population) is mainly due to the high rates among younger doctors.[20]

The final set of comparisons in this study, of normal early retirements at ages 60–64, was made because of the anticipated effects of pension benefit regulations on ill-health retirements at these ages. It is suggested that at least some of the excess number of normal retirements among male and female anaesthetists were a result of ill health. They may also reflect greater stress and fatigue, together with less job satisfaction, since anaesthetists have more rigid timetables and less sustained contact with patients than others. They also have less opportunity to change work patterns in late career.

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Lung water during the puerperium

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Summary

An alteration in pulmonary water balance might be expected during pregnancy and the puerperium, as a result of the physiological adaptations to pregnancy. A preliminary investigation of this hypothesis was conducted using a radiological method of lung water measurement in 20 normal primigravidae during the early puerperium. Evidence of interstitial lung water was found in seven of the 20 mothers. There was a significant correlation with plasma volume ($p < 0.05$) and fluid turnover ($p < 0.05$) in the group with excess lung water. The implications of increased interstitial lung water and its possible causes during pregnancy are discussed.

Key words

Pregnancy.
Lung; water.

Pulmonary oedema is a rare occurrence during pregnancy. When it does occur the cause may be obvious, for example, pre-existing cardiac disease or the aspiration of gastric contents. The aetiology is not always so clear when, for example, pulmonary oedema follows the use of β_2 sympathomimetics or complicates pre-eclampsia. The physiology of lung water balance and the mechanisms which protect against pulmonary oedema are still incompletely, although more fully understood. The effect of pregnancy on the way in which lungs handle water is not known but it may be relevant to the development of pulmonary oedema during pregnancy and the puerperium. Similarly, modern obstetric and anaesthetic practice may have hitherto unexpected effects on lung water; this has not been investigated, although the possibility has been discussed elsewhere.[1] The aim of the present study was to determine whether there was evidence of increased lung water during the puerperium in a group of normal primigravidae.

Method

Twenty normal primigravidae (mean age 25.4 years, range 19–30 years) were recruited at the antenatal clinic. Informed consent was obtained for the study which had the approval of the local ethical committee.

At 38 weeks gestation, measurements were made of plasma volume by Evans blue dilution, using the method of Hytten and Paintin[2] but with the subjects lying in the left lateral position,

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of total body water by D_2O dilution[3] and of haemoglobin and serum albumin. The study did not affect the management of labour or delivery, nor was any restriction placed on the prescription of intravenous fluids. All patients received Syntometrine 1 ml (ergometrine 0.5 mg and oxytocin 5 units) prior to delivery of the placenta. A detailed fluid balance was kept during labour and the puerperium.

Two chest radiographs were taken of each patient, one in the early puerperium and one at or after the 6-week postnatal visit, so that each patient provided her own control. The first film was taken as soon as reasonably possible after delivery, but because this involved travelling to the X ray department of an adjacent hospital which was not available at weekends, this interval was unavoidably variable. The films were taken postero-anteriorly in full inspiration with low voltage in order to show lung detail. The same X ray tube and film cassette was used in each case; tube–film distance was 1.8 m and exposure time was always less than 0.03 seconds.

On the sixth day post partum, the measurements made antenatally were repeated and the fluid balance completed. In order to obtain an expression of fluid turnover, the total fluid input and output during labour and subsequently until the first chest X ray were summed and divided by the number of hours elapsed.

At the end of the study, the pairs of radiographs were coded and presented to the radiologist (A.F.M.) with the dates obscured. They were scored for signs of increased lung water using the method of Pistolesi and Giuntini,[4] modified to exclude signs which could be affected by extrapulmonary tissue density or which were dependent on the increased pulmonary blood flow of pregnancy. The modified scoring system is illustrated in Table 1. In order to minimise false positive results, a minimum score of 15 points was taken to indicate increased lung water. All radiographs with a score of 15 points or more had at least four of the features individually listed in Table 1. The patients were then divided into two groups on the basis of the radiological findings. Statistical analysis was with Student's *t*-test for unpaired observations, or a test of the difference between two proportions (Z), as appropriate.

Table 1. Chest X ray score sheet.

	Score	Film 1	Film 2
Chest X ray findings			
Hila blurred	3, 6, 9		
Kerley's A lines	4, 8		
B lines	4, 8		
C lines	4, 8		
Micronoduli	4, 8		
Widening of scissurae	4, 8		
Peribronchial and perivascular cuffs	4, 8, 12		
Extensive perihilar haze	4, 8, 12		
Subpleural effusion	5, 10		
Additional findings of rare occurrence			
Perilobular lines and rosettes	4, 8		
Unusual linear shadows	4, 8		

Results

Seven of the 20 patients had radiological evidence of interstitial pulmonary oedema during the early puerperium, as indicated by a score of 15 or more points on the postdelivery chest X ray. The results for the group with normal postdelivery radiographs (group A) are shown in Table 2 and for those showing interstitial oedema (group B) in Table 3.

There was no difference between the two groups in terms of total body water, serum albumin or haemoglobin concentration. Plasma volume was higher in group B both antenatally ($p < 0.05$) and postnatally ($p < 0.05$), as was fluid turnover ($p < 0.05$). Mean fluid balance (adjusted by 1 litre for insensible loss in labour and 0.5 litre for each day thereafter) was more positive in group B but this was not significant.

Five of the seven patients in group B were given oxytocin in the first stage of labour compared with four out of 13 in group A. Five in group B received intravenous fluids compared with eight out of 13 in group A, and two in group B had labour induced compared with three out of 13 in group A. These proportions were not significantly different. There was no significant difference in the interval between delivery and chest X ray, in duration of labour or in age between the two groups. The mean age of patients in group A was 25.1 years (SD 3.2) and in group B 27.7 years (SD 3.4).

Discussion

A variety of methods of lung water detection and measurement exist but, unfortunately, many

Table 2. Summary of results from patients in group A.

CXR score	Antenatal* water (%)	Postnatal† water (%)	Antenatal albumin (g/litre)	Postnatal albumin (g/litre)	Antenatal plasma volume (litres)	Postnatal plasma volume (litres)	Fluid balance to CXR (litres)	Fluid turnover (litres)	Fluid in labour (litres)	Post partum day of CXR	Duration of labour (hours)	Antenatal haemoglobin (g/dl)	Postnatal haemoglobin (g/dl)	Oxytocin in first stage (units)	Total oxytocin (units)
0	63	60	30.0	33.0	2.96	2.34	—	—	0	4	7.0	11.5	11.3	0	5.000
0	66	60	35.5	38.5	3.33	2.95	+1.200	0.139	1.100	5	12.0	12.2	11.6	0	5.000
0	65	62	37.5	38.0	3.68	—	+0.985	0.099	0	2	12.0	12.9	13.0	0	5.000
6	55	53	31.2	32.5	4.01	3.26	+1.225	0.117	2.825	4	10.0	11.3	11.1	10.406	15.406
0	65	60	32.0	—	2.55	2.34	−0.460	0.046	0	1	7.0	12.5	11.1	0	5.000
0	58	58	32.0	—	3.45	3.38	+2.150	0.158	1.360	2	9.0	—	10.0	0	5.000
0	66	68	34.5	39.5	3.97	3.31	+2.275	0.219	0.215	5	7.5	11.4	11.7	0	5.000
10	60	53	37.0	33.0	4.12	3.45	+0.100	0.176	1.060	3	7.5	12.6	12.2	0.700	5.700
0	65	62	31.5	32.5	3.22	2.49	−0.090	0.101	2.800	1	6.5	10.0	10.8	0.190	5.190
0	46	45	41.5	42.0	3.66	3.05	−0.430	0.176	0.030	5	7.0	11.3	11.8	0	5.000
0	64	65	31.5	33.6	3.00	2.87	−0.200	0.067	0.300	1	4.5	12.9	10.5	0	5.000
0	60	—	24.5	—	2.82	—	−0.870	0.062	0	2	5.5	10.7	—	0	5.000
0	68	63	31.5	31	3.55	3.08	—	—	1.000	1	7.0	11.4	9.4	3.700	8.700
n‡	13	12	13	10	13	11	11	11	13	13	13	12	12	13	13
Mean	61.6	59.1	30.1	35.4	3.41	2.96	+0.437	0.124	0.6285	2.8	7.9	11.7	11.2	1.154	6.154
(SD)	(6.0)	(6.2)	(4.2)	(3.8)	(0.487)	(0.406)	(1.067)	(0.055)	(0.8289)	(1.6)	(2.27)	(0.90)	(0.97)	(2.961)	(2.961)

* Antenatal measurements made at 38 weeks gestation.
† Postnatal measurements made on the sixth day post partum.
‡ n = Number of results.

Table 3. Summary of results from patients in group B.

CXR score	Antenatal* water (%)	Postnatal† water (%)	Antenatal albumin (g/litre)	Postnatal albumin (g/litre)	Antenatal plasma volume (litres)	Postnatal plasma volume (litres)	Fluid balance to CXR (litres)	Fluid turnover (litres)	Fluid in labour (litres)	Post partum day of CXR	Duration of labour (hours)	Antenatal haemoglobin (g/dl)	Postnatal haemoglobin (g/dl)	Oxytocin in first stage (units)	Total oxytocin (units)
15	62	58	31.0	36.5	4.23	3.38	+3.360	0.195	4.250	3	19.5	10.9	10.9	8.019	13.019
18	68	63	30.5	36.0	3.31	3.30	+4.360	0.194	0.030	6	5	12.5	11.1	0	5.000
19	62	65	35.0	33.0	—	—	+1.200	0.110	1.605	2	14.5	12.2	—	9.790	14.790
15	60	58	34.0	41.0	—	2.88	−2.195	0.259	0.060	4	5	13.6	14.0	0	5.000
15	64	65	31.9	29.6	4.62	4.18	+4.310	0.132	1.850	5	16.5	11.0	10.0	0.240	5.240
25	59	55	31.0	36.0	4.85	3.86	+0.600	0.244	0.975	2	4	10.1	10.6	3.630	8.630
23	63	65	30.6	31.2	3.47	3.28	+0.235	0.139	1.680	1	10	12.0	9.4	12.640	17.640
n‡	7	7	7	7	5	6	7	7	7	7	7	7	6	7	7
Mean	62.9	61.3	32.0	34.8	4.10	3.48	+1.696	0.182	1.493	3.3	10.6	11.8	11.0	4.903	9.903
(SD)	(2.9)	(4.2)	(1.8)	(3.8)	(0.683)	(0.463)	(2.430)	(0.057)	(1.428)	(1.8)	(6.27)	(1.17)	(1.6)	(5.242)	(5.242)

* Antenatal measurements made at 38 weeks gestation.
† Postnatal measurements made on the sixth day post partum.
‡ n = Number of results.

are unsuitable for use during pregnancy or the puerperium, for example, those using radioactive tracers.[5,6] The thermodye dilution method,[7] which involves femoral artery cannulation, was considered to be too invasive, while a less invasive method based on magnetic resonance imaging[8] is not sufficiently sensitive, since it does not distinguish between extravascular and intravascular water. The choice of a radiological method imposed two restrictions on the study, namely, that it could not be used until after delivery and that measurement was qualitative rather than quantitative.

The chest radiograph, when taken under carefully defined conditions, provides the most sensitive non invasive means of detecting small increases in lung water.[9] Studies have shown that the radiograph becomes abnormal when lung water increases by 30–80%.[6,10] Attempts to translate this qualitative information into quantitative data have been devised, usually by means of ascribing scores to various radiological signs. The scoring system described by Pistolesi and Giuntini[4] was derived from the radiographs of patients in cardiac failure, and includes the signs of venous engorgement which precede the development of pulmonary oedema. This may be one reason for these authors' observation that the radiograph became abnormal before any increase in lung water was measurable by indicator dilution. The presence of dilated pulmonary vessels during pregnancy or the puerperium does not usually imply cardiac failure, however, but rather reflects the physiological increase in blood volume. The scoring system was modified in the present study to include only signs which directly reflect the presence of increased extravascular water. The anatomical basis for these signs, for example, peribronchial cuffing and Kerley's lines, has been substantiated histologically by a postmortem study of human lungs.[11]

The radiological detection of interstitial pulmonary oedema is most sensitive when a normal control film of the same individual is available for comparison.[12] Early in the study it became apparent that if the same exposure was given to the control film taken 6 weeks post partum as to the earlier film, the blackening was so much greater that comparison between the two was impossible. This difference, caused by the extra fat and water content of the subcutaneous tissues during pregnancy, may have obscured a real difference in lung field density, but because of the altered technique it was considered that lung field density could not be ascribed a score. All the radiographs with a score of 15 or more points had at least four of the features listed in Table 1. On this basis it seemed reasonable to interpret a score of 15 points or more as indicative of a real increase in interstitial lung water, although no quantitative value for lung water could be derived from the radiographic score because of these modifications.

Radiological evidence of interstitial pulmonary oedema was found in seven of the 20 subjects. This finding poses two questions: namely, what might have caused it and does it matter? The present study could not set out to determine the aetiology of the oedema, but the correlations which emerged may indicate some possibilities. It is possible that the pulmonary changes are part of the general increase in connective tissue water which occurs during pregnancy.[13,14] No correlation was found, however, with total body water. Alternatively, pulmonary fluid filtration may have been increased and there are grounds for predicting that this might occur as a result of some of the physiological adaptations to pregnancy. This hypothesis has been explored more fully elsewhere.[1] Fluid is constantly filtered from the pulmonary vascular bed. The forces that influence this are traditionally described by the Starling relation,[15,16] although it may not completely encompass the situation;[17] other factors such as pulmonary blood flow[18] and plasma protein flux[19] are also important. The relation takes the form

$$Q_f = Lp \cdot A[(P_{mv} - P_{pmv}) - \sigma(\pi_{mv} - \pi_{pmv})]$$

where Q_f is the net transvascular fluid flow, Lp is the membrane hydraulic conductivity per unit area, A is the exchanging surface area, P_{mv} is the microvascular hydrostatic pressure, P_{pmv} is the interstitial hydrostatic pressure, σ is the Staverman reflexion coefficient, π_{mv} is the plasma oncotic pressure and π_{pmv} is the interstitial oncotic pressure.

A fall in plasma oncotic pressure (which is closely related to serum albumin) favours an increase in filtration. The reduced values of plasma albumin expected during the puerperium[20,21] were observed in this study, but there was no difference between the two groups. Plasma volume was significantly greater both antenatally and postnatally in the patients with excess lung water. An increase in plasma volume might increase either the microvascular hydrostatic pressure or the membrane surface area or both, particularly in combination with a raised cardiac output. The question of whether the increase in cardiac output is sustained until delivery appears to have become controversial again,[22] but cardiac output is certainly increased during the early puerperium. In a study using sheep, when cardiac output and circulating volume were increased by a transfusion of whole blood, pulmonary fluid filtration increased.[23]

The other significant correlation in the present study was with fluid turnover. Extravascular extracellular water increases during pregnancy. Water excretion is impaired at term[24] and the puerperal diuresis may not begin until the fourth postpartum day,[25] so that water content may increase further during the 48 hours following delivery. The circulating volume may be expanded by water introduced into the circulation via the gut, directly by infusion or from the extravascular spaces, particularly when the diuresis begins. The expression of fluid turnover is therefore a better measure of water which has passed through the circulation from whatever source than input or output alone. Oxytocin, which has a significant antidiuretic effect even in very low dosage,[26] did not appear to affect the incidence of increased lung water.

In order to consider the functional implications of interstitial pulmonary oedema it is necessary to understand the importance of the pulmonary interstitial compartment in protecting the alveoli. There is normally a small net loss of fluid from the pulmonary microvessels.[27] This filtrate drains from the alveolar interstitium into the perivascular peribronchial lymphatics and their surrounding loose connective tissue and returns to the venous circulation via the thoracic duct. Influxes of filtrate which may occur following rises in vascular pressure,[28] rapid infusions of intravenous fluid[29] or increases in vascular permeability[30] are at first absorbed by the interstitium. Once the capacity of the interstitial compartment has been exceeded, however, and if the lymphatics cannot keep pace with the rate of filtration, alveolar flooding becomes inevitable.[31] This function of the interstitium is more complex than that of a simple reservoir, since alterations in interstitial fluid and plasma protein content in turn alter the interstitial hydrostatic[32] and oncotic pressures[33] and the hydraulic conductivity of the endothelial barrier.[33,34] Furthermore, lymphatic drainage is impeded by rises in systemic venous pressure.[29] This complexity is reflected in the fact that intravascular hydrostatic pressure and plasma oncotic pressures are unreliable predictors of pulmonary oedema when considered in isolation.[35,36]

If the pulmonary interstitium is already hydrated to a greater extent than normal, the protection which it can provide against further increases in lung water is reduced. In the majority of cases, this presumably does not matter. However, there are situations where the obstetric patient appears to be at particular risk of developing pulmonary oedema, for example, when β_2 sympathomimetics are used to suppress premature labour. This response to β_2 sympathomimetics appears to be unique to obstetric patients and is a surprisingly frequent phenomenon in a young and healthy population (5% in one prospective study),[37] but one which might be explained more easily by the presence of preexisting interstitial oedema.

Conclusions

The findings of this study suggest that lung water may increase as a result of pregnancy and/or labour. In the straightforward situation this small increase is clearly not important; normal pregnancy does not result in pulmonary oedema. The deterioration in mitral stenosis during pregnancy and parturition is, however, well recognised.[38] If the pulmonary interstititium contains extra water either because of increased fluid traffic or as part of the generally increased hydration of ground substance, the functional implication is presumably the same, namely, that the interstitial safety margin is reduced. The important question, therefore, is whether some obstetric patients are more susceptible to pulmonary oedema than their nonobstetric counterparts. If this is so, then rises in pulmonary vascular pressures and cardiac output (which may occur during tracheal intubation or follow the administration of ergometrine[39]) or large intravenous fluid loads, may be more hazardous than might be expected in this young population. An explanation of such problems as the apparent increased severity of Mendelson's syndrome in obstetric situations and the occurrence of pulmonary oedema in pregnant patients receiving β_2 sympathomimetics, may be related to such an alteration in lung water balance. These questions cannot be answered until the effects of pregnancy on lung water handling have been fully investigated.

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Anaesthesia, 1987, Volume 42, pages 148–150

The effect of pregnancy on pulmonary nitrogen washout

A study of pre-oxygenation

F. BYRNE, A. ODURO-DOMINAH AND R. KIPLING

Summary

Pre-oxygenation times were studied in 30 fit pregnant women during the course of their pregnancy. The mothers were studied in the supine position with a left lateral tilt and breathing through a standard Magill system with an oxygen flow rate of 8 litres/minute. Between 13–26 weeks gestation and 26–42 weeks the times needed to achieve an end-tidal nitrogen of 2% were 104 seconds (SD 30) and 80 seconds (*SD 20*), *respectively. A control group of young women of similar age and weight needed 130 seconds (SD 30) to achieve the same end-tidal nitrogen concentration. A gas-tight mask was found to be essential to achieve effective denitrogenation (end-tidal nitrogen concentration of 2%).*

Key words

Anaesthesia; obstetric.
Oxygen; delivery systems.

The confidential report on maternal mortality of 1976–78[1] found 40 deaths associated with anaesthesia. Sixteen of these were the result of difficulties in establishing tracheal intubation during the induction of general anaesthesia. Adequate pre-oxygenation in such patients is therefore important. Archer and Marx[2] confirmed the existence of increased oxygen consumption in pregnant women, especially at term. Progressive reduction of the functional residual capacity (FRC) during pregnancy has been consistently reported.[3–6] However, studies of the optimum duration of pre-oxygenation have all been performed on healthy non-pregnant patients and therefore the recommendations made may not necessarily apply to pregnant patients, especially at term.

Methods

Sixty ASA 1 women aged between 20 and 36 years were studied. Thirty of these were pregnant women and formed the study group, while the remainder formed the control group.

The study group patients were seen at their first antenatal clinic visit and informed consent was obtained. Measurements were made, in the Pulmonary Function Clinic, at the following times: first visit 13–26 weeks, second visit 30–34 weeks and third visit 38–42 weeks. The control group was studied on one occasion only. Measurements in all subjects were made with a left lateral tilt and with the head raised on one pillow.

A standard Magill system with a close-fitting mask was used and a sampling needle inserted into the mask *via* the angle piece. The subject's tidal volume was measured first, using a Morgan dry spirometer. She then breathed 100% oxygen, supplied at a flow rate of 8 litres/minute, from the Magill breathing system, starting with the

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reservoir bag full.[7] The time taken to achieve an end-tidal nitrogen concentration of 2% was noted. There was a rest period of 5 minutes before the same measurement was repeated. The mask was held on the subject's face to ensure a gas-tight seal.

Analysis of the expired mixture was made using the 505 Medscience Nitranalyser with a response time of 0.05 seconds. This equipment was calibrated with room air according to the manufacturer's instructions. The output was recorded on a J.J. Lloyd Recorder Chart. The records were analysed to determine the times needed to reach an end-tidal nitrogen concentration of 2% for each group. Analysis of variance was used to compare the results.

Results

Although tidal volume was measured, no attempt was made to control it or the respiratory frequency. The results are shown in Table 1. There was a statistically significant difference in the pre-oxygenation times between the first half of pregnancy (up to and including the 25th week, group 1) and the second half of pregnancy (from the 26th week to term, group 2). The patients were studied on three occasions, namely at 13–26 weeks, 30–34 weeks and 38–42 weeks, but the last two gave similar results; these were therefore combined to form group 2. Group 1 showed a mean pre-oxygenation time of 104 seconds (SD 30) and group 2 a mean of 80 seconds (SD 20). Thus, 95% of group 2 achieved an end-tidal nitrogen concentration of 2% within 2 minutes. These results were highly significantly different ($p < 0.001$). The mean time to achieve an end-tidal nitrogen concentration of 2% in the control group was 130 seconds (SD 30). This time was highly significantly longer than for the other two groups. The increase in resting expired minute volume in group 2 (Table 1) is almost certainly due to the physiological effects of advancing pregnancy.

Discussion

Various recommendations have been made concerning the duration of pre-oxygenation for adult non-pregnant patients.[7] Suggested times range from 2–10 minutes, but none of these relates specifically to pregnant women. During breathing 100% oxygen, the rate of denitrogenation is influenced by the degree of ventilation and the functional residual capacity (FRC).

The present study shows a progressive reduction in the pre-oxygenation times with advancing pregnancy. A combination of factors are responsible for this. An increase in minute volume is a physiological effect of advancing pregnancy, but this effect will be modified by changes in the distribution and pattern of ventilation. There is an 18% reduction in functional residual capacity[3] during pregnancy, which reaches statistical significance at 28–30 or 38–40 weeks of gestation,[6] depending on whether postpartum or early pregnancy functional residual capacity is used as a control. The result of the reduced functional residual capacity is to diminish the oxygen reserve within the lung, so that the parturient patient is able to withstand only very short periods of apnoea. Our results indicate that, at term, 95% of the patients in the study achieved an end-tidal nitrogen concentration of 2% within 2 minutes (80 seconds, SD 20) breathing 100% oxygen from a Magill system, starting with the reservoir bag full. An airtight seal was found to be essential. The control group's time of 130 seconds (SD 30) provides a useful comparison with that of the study group.

Table 1. Patient data.

	Group 1 (13–26 weeks)	Group 2 (26–42 weeks)	Control
Mean height, cm (SD)	162.6 (5.4)	162.6 (5.4)	159.5 (7.0)
Mean age years (SD)	25 (3.8)	25 (3.8)	25.5 (3.2)
Mean expired minute, volume, litres (SD)	9.79 (1.7)	11.9 (2.8)	7.75 (1.7)
Mean Time to end-tidal nitrogen of 2% seconds (SD)	104 (30)	80 (20)	130 (30)

Although the two groups were of similar age, the control group took an average of 50 seconds longer to achieve an end-tidal nitrogen concentration of 2%. Their time agrees closely with recommendations for non-pregnant preoxygenation times.

The fact that an end-tidal nitrogen concentration of 2% could be achieved within 2 minutes is of particular importance in the context of Caesarean section, especially when there is a need for rapid induction of anaesthesia because of fetal distress.

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A new percutaneous infraclavicular approach to the axillary vein

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Summary

A new percutaneous infraclavicular approach for cannulating the axillary vein is described. The technique was devised using surface landmarks established by cadaver dissection. This is a relatively safe procedure with no risk of pneumothorax, provided that the tip of the needle remains inferior to the clavicle.

Key words

Veins; cannulation.

Direct percutaneous cannulation of the large central veins is most commonly achieved using either the internal jugular or the subclavian vein.[1] However, these techniques are not without hazard. Indeed, in view of the proximity of the thoracic inlet, these two routes probably have the most potential for serious acute complications,[1–9] particularly pneumothorax and haemothorax. While a pneumothorax can be effectively treated by inserting a chest drain, a vascular laceration (artery or vein) sufficient to cause a haemothorax usually requires surgical exploration.

The more distal axillary approach to the axillary vein[10–13] avoids these particular complications, although there is a risk of damaging the medial cutaneous nerve of the arm, since this nerve lies immediately medial to the vein in the axilla.[10] Access to the central veins via the external jugular vein is sufficiently unreliable for it not to be considered a route of first choice, although it is being increasingly used surgically for tunnelled Hickman catheters,[14] as well as for central venous pressure monitoring.[15] There are also complications with this approach.[16,17] Access via the femoral vein is complicated by the potential for thrombo-embolism and therefore only tends to be used when the usual central venous access routes are particularly difficult. The basilic vein can only be used with long narrow catheters, which are sometimes very difficult to thread centrally.

There are, therefore, sufficient problems associated with the commonly used routes for cannulation of the large central veins, to stimulate a continued search for safer routes of access. One approach is to consider different ways of using existing routes; for example, the 'half-way' catheter concept,[18–21] whereby a peripheral arm-catheter is advanced only as far as the axillary vein. Although not suitable for central venous pressure monitoring, such a technique is satisfactory for intravenous feeding. Alternatively, new routes of access can be sought. To this end, the infraclavicular region was explored in the dissecting room (adult female cadaver) with a view to establishing suitable surface landmarks to facilitate an infraclavicular approach to the axillary vein.

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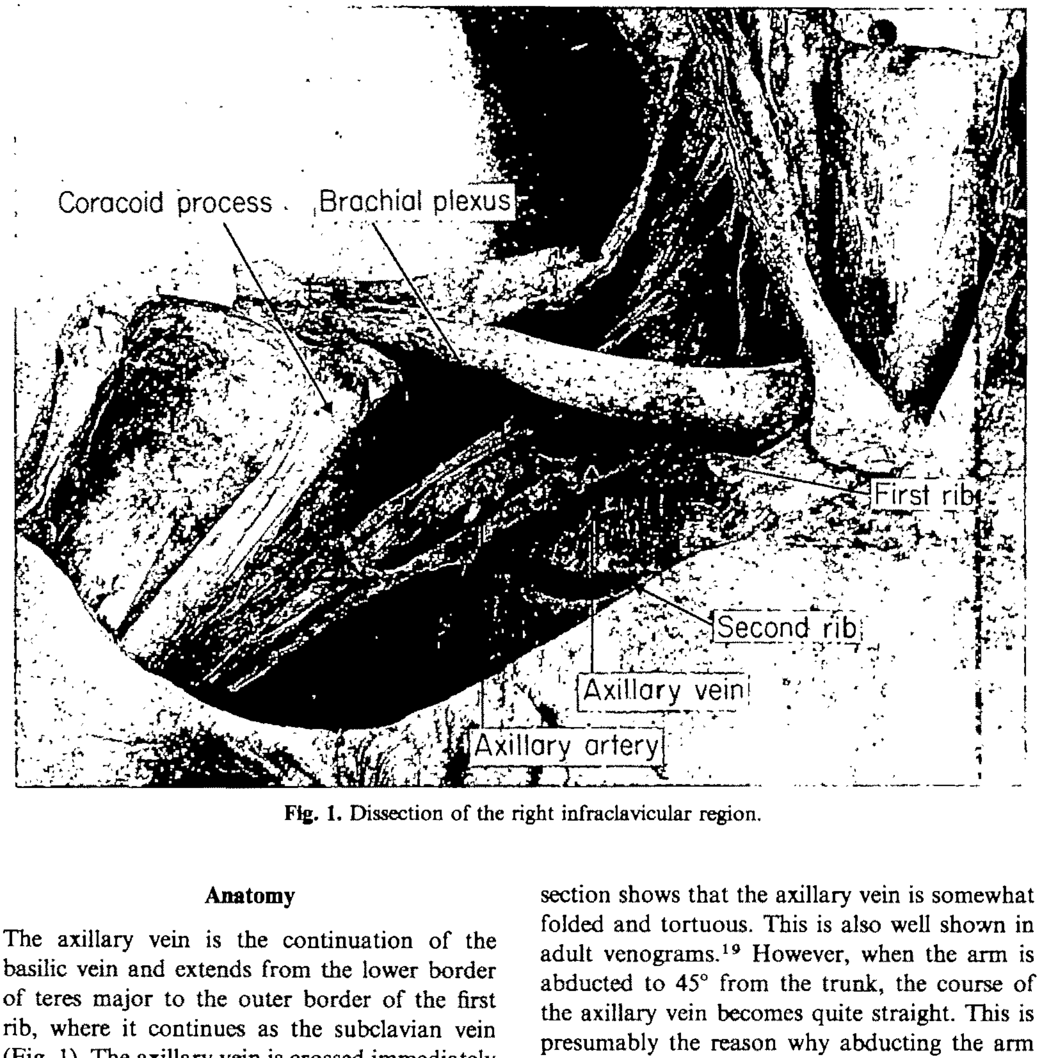

Fig. 1. Dissection of the right infraclavicular region.

Anatomy

The axillary vein is the continuation of the basilic vein and extends from the lower border of teres major to the outer border of the first rib, where it continues as the subclavian vein (Fig. 1). The axillary vein is crossed immediately anteriorly by pectoralis minor, which divides the vein topographically into three parts, namely proximal, posterior and distal to pectoralis minor.[22] The medial border of pectoralis minor extends from the coracoid process along a line roughly at 45° to the long axis of the body (Fig. 2). However, there is some variation, since although this muscle is usually inserted into the third, fourth and fifth ribs, it not uncommonly arises from the second, third and fourth ribs.[22]

The axillary vein is a large vein, with a mean proximal diameter of 16 mm in adults during a Valsalva manoeuvre;[19] this compares well with a value of 19 mm for the proximal part of the subclavian vein under similar conditions.[19] With the arm adducted to the side of the trunk, dissection shows that the axillary vein is somewhat folded and tortuous. This is also well shown in adult venograms.[19] However, when the arm is abducted to 45° from the trunk, the course of the axillary vein becomes quite straight. This is presumably the reason why abducting the arm often facilitates central cannulation via the basilic vein. The axillary artery is closely related to the axillary vein. It is posterolateral to the vein as it passes over the first rib and becomes lateral to the vein as it enters the upper arm.

Surface landmarks

The dissection study suggested the following medial and lateral surface landmarks for the infraclavicular course of the axillary vein in an adult; these are shown in Fig. 2. The lateral landmark (A) is three finger breadths (5 cm) below the inferior aspect of the coracoid process. The coracoid process is a particularly suitable reference point, since it is easily palpable even in obese patients. The medial landmark (B) is that

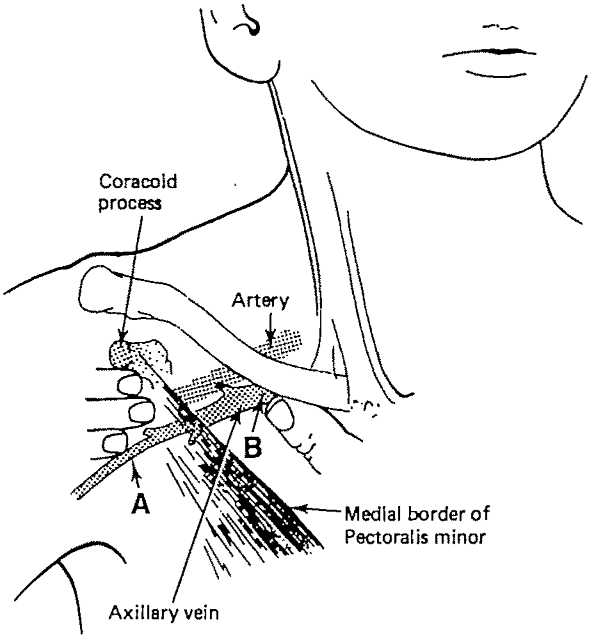

Fig. 2. The lateral (A) and medial (B) surface landmarks of the axillary vein.

point below the medial end of the clavicle where the space between the clavicle and the thorax just becomes palpable. This is approximately at the junction of the medial quarter and the lateral three-quarters of the clavicle.

Method for cannulating the axillary vein

Position the patient as for the subclavian approach[1] (supine and approximately 15° Trendelenberg).

Abduct the arm to 45° from the trunk, keeping the arm straight.

Mark the lateral (A) and medial (B) surface landmarks as shown in Fig. 2. Mark the position of the medial border of pectoralis minor where it cuts the line AB.

Under the usual sterile conditions, enter the skin on the line AB and lateral to the medial border of pectoralis minor, directing the needle medially in the anteroposterior (A–P) plane which passes through the line AB. Aim to enter the vein between pectoralis minor and point B. Should there be any resistance at all while advancing the needle, the needle should be withdrawn and redirected in an A–P plane, parallel to the line AB.

Patients

This method for cannulating the axillary vein has so far been used successfully and easily in 13 out of 14 patients, using a Seldinger technique (Leder-cath; Vygon). There were no arterial punctures or other complications. The one failure to locate the vein using this approach was in a grossly obese patient, in whom a standard subclavian approach eventually had to be used.

Discussion

The particular merit of this approach to the axillary vein is that it combines cannulation of a large vein well away from the thoracic inlet, with the convenience of easy and clean skin fixation in the infraclavicular region. Experience so far in adults (both male and female) suggests that the given surface landmarks are reliable, in spite of their being determined only in one female cadaver. Cannulation is facilitated by the fact that when the arm is abducted to 45° from the trunk the vein is quite straight, allowing the needle to be directed along the line of the vein.

Although the risk of pneumothorax with this technique would seem to be minimal (provided that the tip of the needle remains inferior to the clavicle), there is a risk of inadvertent puncture of the axillary artery, since the artery cannot generally be felt infraclavicularly. Initial probing with a small-gauge needle to locate the vein is therefore recommended.

The results so far are encouraging, and demonstrate that cannulation of the axillary vein via an infraclavicular approach is a practical proposition.

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Myocardial ischaemia during non-cardiac surgery†

A comparison of different lead systems using computerised ST segment analysis

R. M. GRIFFIN AND J. A. KAPLAN

Summary

Computerised ST segment analysis was used to compare the frequency of ischaemia occurring in electrocardiographic leads II, V_5, CS_5 *and* CB_5. *Three out of 15 patients with ischaemic heart disease developed ischaemic changes, which were evident in all four leads in each patient. A single bipolar lead may be substituted for a true* V_5 *lead when monitoring patients at increased risk of developing myocardial ischaemia. ST segment analysis facilitates the early diagnosis of peri-operative myocardial ischaemia, which may otherwise be missed on the standard electrocardiogram.*

Key words

Complications; myocardial ischaemia.
Monitoring techniques; electrocardiogram.

Myocardial ischaemia may occur in patients with a known history of ischaemic heart disease (IHD) during both cardiac[1,2] and non-cardiac surgery.[3,4] At present, the electrocardiogram is usually monitored at induction and intra-operatively to detect any ischaemic episodes. However, the value of this monitoring depends upon which electrocardiographic lead is selected. The precordial V_5 lead has been recommended for use during anaesthesia,[5] but this requires a five-lead system which is not usually available in the operating theatre. In this situation, a bipolar lead may be substituted, using a three-lead system. Several bipolar leads have been described for use during exercise stress testing,[6,7] but no previous comparison of multiple leads has been made during and after anaesthesia, using computerised ST segment analysis.

Methods

The study was approved by the Research Committee of the Mount Sinai Hospital. Written, informed consent was obtained from 15 patients with IHD. Diagnosis of IHD was based on a clear history of effort-related chest pain, previous history of myocardial infarction (MI) or electrocardiographic changes of myocardial ischaemia. Patients with conduction defects or left ventricular hypertrophy with strain, which may produce difficulty in the interpretation of ST segment depression, were excluded. There was no restriction on age, type of surgery or preoperative drug therapy. In addition to this group of patients, seven healthy patients, free of symptoms and signs of IHD, were studied as a control group to determine the incidence of false

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positive ST segment changes. Premedication, anaesthesia and monitoring were chosen by the anaesthetist in charge of the case. No attempt was made to influence the type of monitoring or conduct of anaesthesia. However, the anaesthetist was informed of any ischaemia not apparent on the standard ECG, so that appropriate treatment might be given. Prior to the induction of anaesthesia, five electrodes were placed on the patient to monitor leads II, V_5 and modified leads CS_5 and CB_5. Two electrodes were positioned in the V_5 position, one electrode on the left hip, one electrode under the mid-point of the right clavicle and one electrode posteriorly in the middle of the scapula (Fig. 1). The anaesthetist was advised to select an ECG lead for each patient according to his or her usual practice. A modified Hewlett–Packard 2008A module allowed the selection and continuous display of lead II and any one of the leads V_5, CS_5 or CB_5. A Quinton 741 ECG data computer was used for ST segment analysis of the electrocardiogram (Fig. 2). The computer uses an averaging memory to develop an ECG complex that represents the average of the previous 20 beats. This complex is then electronically sampled to provide four ST parameters: ST level, slope, index and integral. Digital values of heart rate averaged over 15 seconds, ST level and one other ST parameter are continuously displayed. In addition, the ST segment parameters are displayed as a histogram at the end of each 20 beats. A written record of ST segment parameters for each lead was obtained at 5-minute intervals, or more frequently during periods of critical anaesthetic or surgical interventions. Ischaemia was defined as 1 mm or more of downsloping or horizontal ST segment depression, greater than 2 mm of upsloping ST segment depression, measured 0.06 seconds from the J-point and ST elevation greater than 1 mm. The ECG was monitored during induction and maintenance of anaesthesia and until discharge of the patient from the recovery room. Postoperatively, all patients with IHD had serial 12-lead ECGs in the recovery room and on days 1, 3 and 5, and blood taken for serum creatine phosphokinase–myocardial band levels (CPK-MB).

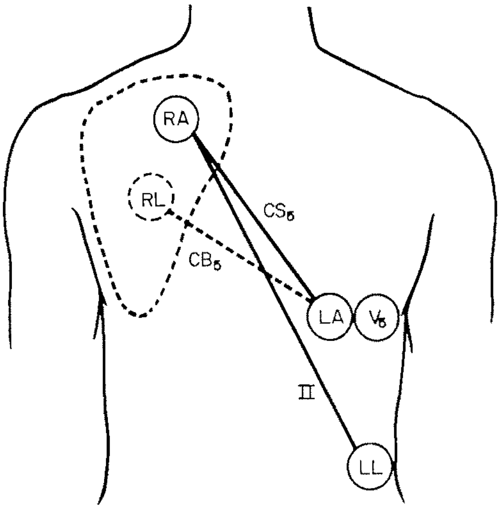

Fig. 1. Position of electrodes. RA, right arm; RL, right leg; LA, left arm; LL, left leg. For lead CB_5, RA and RL leads were transposed.

Results

Details of the patients with IHD and the drugs used for induction and maintenance of anaesthesia are given in Table 1. No patients in the control group developed any electrocardiographic changes of ischaemia. Three of the patients with IHD showed ischaemia on the ECG. Table 2 gives the ST parameters recorded for these patients before and after the development of ischaemia. One patient developed ischaemia

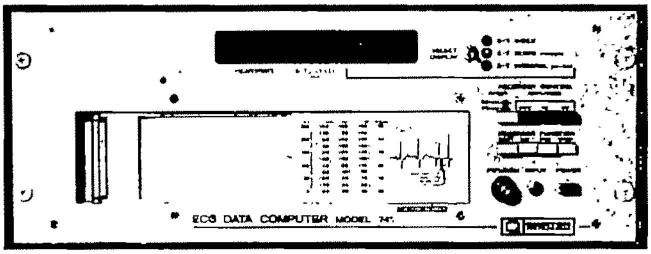

Fig. 2. Front panel of ECG data computer.

Table 1. Individual details of patients, surgery and anaesthesia.

	Age	ASA status	Operation	Induction	Maintenance
F* 1	53	3	Transurethral resection of bladder tumour	Thiopentone, suxamethonium	Isoflurane
M 2	63	3	Aortobifemoral bypass graft	Fentanyl, thiopentone, pancuronium	Halothane
F 3	76	3	Lumbar sympathectomy	Fentanyl, thiopentone, suxamethonium	Fentanyl, curare
M 4	57	3	Biopsy leg tumour	Thiopentone	Enflurane
M 5	57	2	Ventral hernia repair	Thiopentone	Enflurane
M 6	52	3	Gastroenterostomy pancreatic/liver, lymph node biopsy	Thiopentone, suxamethonium	Enflurane, pancuronium
M 7	78	2	Cystoscopy/transurethral resection of prostrate	Spinal tetracaine 14 mg	
F 8	73	3	Femoropopliteal bypass	Thiopentone, fentanyl, suxamethonium	Fentanyl, curare
F 9	84	3	Austin Moore hip prosthesis	Thiopentone, suxamethonium	Isoflurane, pancuronium
F 10	60	3	Anterior resection	Fentanyl, diazepam, suxamethomium	Fentanyl, pancuronium, isoflurane, droperidol
M 11	73	2	Transurethral resection of bladder tumour	Spinal tetracaine 12 mg	Thiopentone, diazepam
F* 12	69	3	Right inguinal herniorrhaphy	Spinal tetracaine 12 mg	
F* 13	92	4	Cholecystectomy choledochojejunostomy	Fentanyl, thiopentone, atracurium	Isoflurane, atracurium
M 14	51	3	Below knee amputation	Thiopentone, fentanyl, atracurium	Isoflurane, atracurium
F 15	73	3	Cholecystectomy	Thiopentone, suxamethonium	Isoflurane, pancuronium

* Patients who developed myocardial ischaemia.
All patients received 30–50% nitrous oxide in oxygen.

Table 2. Mean values of ST segment parameters for each lead in patients with ischaemia.

	II		V_5		CS_5		CB_5	
	C*	I†	C	I	C	I	C	I
ST depression (mm)	0.2	1.0	0.1	1.1	0.4	1.8	0.4	1.4
ST slope (mm/second)	1.3	−2.3	2.6	0	2.3	−2.3	3.6	−3.7
ST index	−0.1	−0.8	0.2	−0.8	−0.2	−1.6	−0.2	−1.7
ST integral (μV/second)	2.0	6.3	1.9	6.8	3.3	11.2	4.1	9.4

* Control value.
† Value during ischaemia.

shortly after administration of a spinal anaesthetic. In the other two patients, myocardial ischaemia developed during recovery from general anaesthesia. In each patient, the ST segment changes were evident in all four leads which were studied (Fig. 3). Postoperative serial ECGs and CPK-MB estimations did not indicate ongoing ischaemia in any patient.

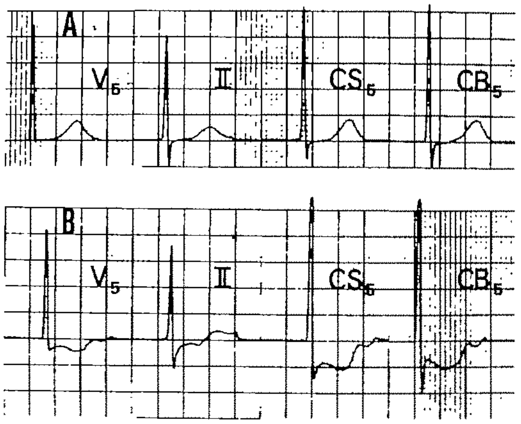

Fig. 3. ECG tracing from patient taken prior to induction (A) and upon awakening (B). ST segment depression is present in all four leads. 1 mV = 20 mm.

Discussion

Peri-operative myocardial ischaemia occurred in 20% of the patients with IHD. Roy *et al.*[3] using multiple exercise leads and ST segment analysis found an incidence of 38% in a similar group of patients. The lower incidence of ischaemia in our patients may be related to the use of specific drugs to maintain cardiovascular stability during anaesthesia. Rao *et al.*[8] have shown that post-operative reinfarction in patients with pre-operative myocardial infarction can be reduced by aggressive, invasive haemodynamic monitoring and early use of appropriate cardiovascular drugs to maintain stability of the circulation. Vasodilator drugs can be used to reduce both preload and afterload and thus produce beneficial effects for the myocardium.[9] Nitroglycerin relieves myocardial ischaemia, probably by both an indirect effect on preload and afterload, thereby reducing left ventricular end diastolic pressure, myocardial wall tension and, consequently, oxygen demand[10,11] and a direct dilating effect on both normal and diseased epicardial coronary arteries.[12,13] The prophylactic use of nitroglycerin infusion during anaesthesia for patients with IHD undergoing coronary artery bypass surgery has recently been investigated.[14]

In the present study, six patients received nitroglycerin peri-operatively and a further six patients were given intravenous lignocaine immediately prior to intubation. Eight patients received both lignocaine and nitroglycerin. Tracheal intubation is a particularly hazardous manoeuvre in patients with IHD.[3] Intravenous lignocaine effectively attenuates the haemodynamic changes associated with tracheal intubation,[15] thereby minimising the risk of myocardial ischaemia occurring at this time. No patients developed myocardial ischaemia during intubation. However, myocardial ischaemia occurred in two patients during recovery from anaesthesia, shortly before extubation. In one patient ischaemia developed shortly after admission to the recovery ward, emphasising the importance of continuing monitoring into the early recovery period. Both episodes were accompanied by severe arterial hypertension and settled with removal of the tracheal tube. The response of these patients confirms that extubation of the trachea may produce adverse haemodynamic changes[16,17] that can precipitate myocardial ischaemia in susceptible patients.

Episodes of myocardial ischaemia were evident in all four leads for each patient. The magnitude of the ventricular deflexions was greatest in leads CB_5 and CS_5, as were changes in certain of the ST segment parameters. Bazaral *et al.*[18] found a good correlation ($r = 0.95$) between the ventricular deflexions of leads CB_5 and V_5 during anaesthesia, although the magnitude of these deflexions was 20% greater in CB_5. Either lead CS_5 or CB_5 is a satisfactory alternative to monitoring a true V_5 lead during anaesthesia for patients who are at high risk of developing myocardial ischaemia. Neither of the two ischaemic episodes which occurred in the operating theatre were noticed by the anaesthetist on the standard ECG monitor. Significant alterations in the ECG complex may be missed, especially if there is no facility to record and compare a baseline ECG complex on the monitor. Use of 'on line' ST segment analysis, especially with a trend facility,[19] may enable more accurate diagnosis of myocardial ischaemia in the peri-operative period.

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Anaesthesia, 1987, Volume 42, pages 160–163

Speed of injection and spread of bupivacaine in the epidural space

R. B. GRIFFITHS, W. A. HORTON, I. G. JONES AND D. BLAKE

Summary

Speed of injection of bupivacaine was assessed in 73 women who received epidural analgesia in labour. Rapid injection was shown to produce quicker onset of epidural block and more reliable perineal anaesthesia than slow injection, but there was no difference in the final extent of the block.

Key words

Anaesthesia: obstetric.
Anaesthetic techniques, regional; epidural.

A fast, rather than a slow, injection of local anaesthetic agent into the epidural space is often used to obtain rapid onset of blockade. A manoeuvre which shortens the delay between injection of local anaesthetic and onset of analgesia, thereby increasing the comfort of the patient, is welcome, provided that it is not accompanied by sudden changes in blood pressure.

This trial was designed to compare rapid and slow injections of bupivacaine into the epidural space. Blockade of temperature sensation was examined as a sensitive and reproducible indicator of the spread of bupivacaine in the epidural space.

Method

Consecutive patients admitted to the labour ward and presented to the authors for epidural analgesia in labour were considered for inclusion in the trial. Patients with pre-eclampsia, breech presentation or multiple pregnancy were not studied. The trial was limited to patients between 151 and 168 cm in height. Each patient was randomly allocated to one of two groups; those in group A received a rapid injection of bupivacaine and those in group B a slow injection.

A further twelve patients were excluded after allocation into a group because of deviation from a standard method of epidural injection. The results of three patients were excluded because the epidural catheter was inserted in a space other than that between the second and third lumbar vertebrae, three because of bloody taps, two because the catheter was directed caudally, two because a contraction occurred during the injection, one because rapid progress of labour indicated a second injection of bupivacaine before assessment of the first was completed and one because the injection was not completed within the time set.

After establishing venous access, though no preload of fluid was given, a standard epidural procedure was used in all cases. With the patient sitting, a 1.6-mm Tuohy needle was inserted in the midline between the second and third lumbar

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vertebrae. The epidural space was identified using a loss of resistance to saline technique. Less than 2 ml of saline was injected after the needle had entered the epidural space. A 16-gauge Portex epidural catheter was passed through the needle with the bevel directed cephalad. Following withdrawal of the needle, 2 cm of catheter was left in the epidural space.

With the patient supine and in a 15° left lateral tilt, an injection of bupivacaine 0.5% with 1:400 000 adrenaline was given through the epidural catheter. All injections were given following the onset of labour, which was indicated by pain or demonstrable uterine contractions. Injections were given between contractions and the blood pressure monitored using a sphygmomanometer. Patients in group A were given the injection of bupivacaine rapidly within 5 seconds. Patients in group B were given the injection slowly over 40 seconds.

Injection of 4 ml of bupivacaine within 5 seconds was achieved using a 5-ml syringe. The injections were not made through a bacterial filter, although the bupivacaine was drawn into the syringe through a filter needle to avoid the injection of glass particles. Using a 20-ml syringe and injecting through a bacterial filter, although more difficult, it is possible to produce a similar jet-like flow from the end of the epidural catheter.

Spread of bupivacaine in the epidural space was monitored using ethyl chloride spray. Block was considered to have occurred when the sensation of cold was markedly reduced or was perceived as warm. Blockade was assessed at 10, 20 and 30 minutes following the injection of bupivacaine.

Dermatome maps vary in their appearance, depending on whether they were constructed by anatomists or clinical physiologists. Many dermatome maps were derived from the clinical observations of Foerster[1] and Keegan.[2] Foerster described a series of patients with nerve root damage and stressed the importance of overlap of dermatomes. Keegan[2] observed patients with intervertebral disc prolapse and maintained that it is possible to identify single nerve root compression. Both authors write as diagnosticians attempting to identify single nerve root damage. The anaesthetist's requirements are quite different; he needs a reliable and reproducible method of assessing blockade of many dermatomes, both to monitor his own practice of spinal and epidural analgesia and also to facilitate meaningful comparison with his colleagues.

Maps of the lumbar and sacral dermatomes are especially varied. It is, however, blockade between the tenth thoracic and the fourth sacral roots that the obstetric anaesthetist most needs to assess. Examination of the various dermatome maps reveals landmarks which are regularly allocated to specific dermatomes. These landmarks, described in Table 1, enabled us to make reliable assessments of blockade and to avoid the inconsistencies of the striped manikins often used.

Table 1. Landmarks used to identify the dermatomes blocked by epidural bupivacaine.

Dermatome level		Skin over the:
Thoracic	2	apex of the axilla
	5	nipples
	7	xiphisternum
	10	umbilicus
	12	lower abdomen above mons pubis
Lumbar	1	inguinal ligament
	2	anterior aspect of the upper thigh
	3	medial aspect of the knee
	4	medial malleolus
	5	dorsum of the foot at the base of the second toe
Sacral	1	lateral aspect of the foot
	2	posterior thigh
	3	medial aspect of the buttock and posterior half of the labia majora
	4	perineal body
	5	coccyx

Using Table 1 and examining temperature sensation, blocked nerve roots were identified. Bilateral blockade of the second, third and fourth sacral dermatomes was regarded as necessary for perineal anaesthesia. Any perineal sensation to ethyl chloride in a patient, indicated that a careful assessment of the sacral dermatomes in that patient needed to be made. The presence of labour pains was recorded at 10-minute intervals.

Results

A total of 73 patients were included in the trial. Thirty-six patients in group A received a fast injection of bupivacaine and 37 patients in group B received a slow injection. The epidural technique was well accepted by all patients. One patient in group A had a decrease in systolic blood pressure of 20 mmHg.

When comparing the two groups, a dermatome was considered blocked only if the block could be demonstrated bilaterally. The numbers of bilaterally blocked segments in each group were compared at 10, 20 and 30 minutes using Student's *t*-test. The numbers of dermatomes blocked at each assessment time are shown in Table 2. At 10 and 20 minutes, significantly more dermatomes were blocked in the fast injection group compared with the slow group ($p < 0.001$ and <0.01, respectively), but at 30 minutes there was no significant difference between the groups with regard to the number of dermatomes blocked. Table 3 shows the numbers of patients in whom perineal blockade was present at 10 and 20 minutes. At the 10-minute assessment, perineal blockade with bilateral block of the second to fourth sacral dermatomes occurred in 13 patients in group A and two patients in group B ($p < 0.001$, Chi squared test). The smaller difference between the two groups at 20 minutes is not significant ($p > 0.1$). At 30 minutes in patients with demonstrated blockade of lumbar and sacral dermatomes, failure to block the first sacral dermatome on the left or right or both sides occurred in 30% of patients in group A and 24% of patients in group B. The resistance of the first sacral root to blockade has been previously reported[3] and has been attributed to its larger diameter.

Table 2. Mean (SEM) number of bilaterally blocked dermatomes in patients following an epidural injection of bupivacaine rapidly (group A) or slowly (group B), assessed at 10, 20 and 30 minutes after injection.

Time after injection (minutes)	Mean (SEM) number of dermatomes blocked Group A (fast injection)	Group B (slow injection)	
10	6.3 (0.62)	3.5 (0.48)	$p < 0.001$
20	10.4 (0.58)	7.7 (0.64)	$p < 0.01$
30	11.3 (0.56)	9.8 (0.69)	NS

Table 3. Number of patients with perineal blockade following an epidural injection of bupivacaine rapidly (group A) or slowly (group B), assessed at 10 and 20 minutes after injection.

Time after injection (minutes)	Number with perineal blockade Group A (rapidinjection)	Group B (slow injection)	
10	13	2	$p < 0.001$
20	25	20	NS

Thirty-four patients in each group had lower abdominal or back pain before the injection of bupivacaine. Twenty-four patients in group A and 17 patients in group B were pain free at 10 minutes although only 4 ml of bupivacaine had been injected. The difference was not significant. The mothers in this trial received their epidural injections early in labour. To assess perineal analgesia rather than blockade of temperature sensation, an examination of patients entering the second stage of labour is needed.

Discussion

When 40 seconds are taken to inject 4 ml of bupivacaine through a Portex epidural catheter, the bupivacaine slowly drips from the catheter tip. However, when the injection is completed in 5 seconds, the bupivacaine leaves the catheter in three vigorous jets. One might expect variations in the effect of these two different methods of injection.

A previous study[4] demonstrated no significant effects due to varying the speed of the epidural injection. In comparison to this study, however, the rates of injection were slow and the volumes of local anaesthetic agent were large. Using large differences in the rates of injection, small volumes of bupivacaine and careful assessment of blockade with ethyl chloride, the effect of the speed of epidural injection has been demonstrated.

The results show a more rapid spread of bupivacaine in the epidural space and earlier establishment of block following a fast injection. The two groups studied show similar spread at 30 minutes, but the earlier, more extensive blockade in group A has clinical implications when attempting to relieve pain in obstetric practice.

A rapid epidural injection of bupivacaine will more reliably produce perineal blockade at 10 minutes. When early onset of analgesia and sacral blockade are required for a controlled or instrumental vaginal delivery, we suggest a rapid injection of local anaesthetic is indicated. A larger dose injected slowly might be expected to produce a similar result in the short term, but the epidural blockade will progress to include more segments than necessary.

Extensive block of temperature sensation was demonstrated in both groups following only 4 ml of bupivacaine with 1:400 000 adrenaline. In four cases, blockade from the seventh thoracic to the fifth sacral dermatomes was seen. This spread does not coincide with the area of surgical analgesia, but it does demonstrate the sensitivity of assessment using ethyl chloride.

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CASE REPORT

Anaphylactoid reaction to alcuronium

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Summary

A 67-year-old woman suffered cardiovascular collapse during induction of anaesthesia. This was later shown to be anaphylactic in origin; the causative agent was alcuronium. In vitro testing showed a highly specific sensitivity to alcuronium with minimal cross-reactivity. Future anaesthesia with decreased risk was thereby assured. The clinical nature of this reaction and a review of the literature implicate the cardiovascular system as the principal target in this type of reaction to alcuronium and suggest that the heart is directly involved.

Key words

Neuromusclar relaxants; alcuronium.
Allergy; anaphylaxis.

Severe anaphylactoid reactions to anaesthetic agents are fortunately uncommon.[1] They usually consist of an acute urticarial skin rash, bronchospasm and a varying degree of cardiovascular instability. Prompt resuscitation is required if disaster is to be avoided. This case report illustrates an atypical presentation of anaphylaxis which was initially clinically misleading and it details the investigations used to help substantiate the diagnosis.

Case history

The patient was a 67-year-old housewife who presented with symptoms of peripheral vascular disease, principally localised to the left foot where there were signs of gangrene in the third and fourth toes. She was scheduled for amputation of these toes.

There were no previous anaesthetics or medical illnesses. Her family history was unremarkable with no abnormal reactions to general anaesthesia. She had stopped smoking 6 weeks previously and was taking dihydrocodeine, 60 mg three times per day. Symptomatic enquiry and physical examination revealed normal cardiovascular and respiratory systems. Preoperative investigations, including a chest X ray, electrocardiogram, full blood count, erythrocyte sedimentation rate, serum electrolytes, protein electrophoresis and antibody screening were normal. Body weight was 61 kg.

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Premedication consisted of intramuscular pethidine 75 mg and promethazine 12.5 mg. Anaesthesia was induced with thiopentone 250 mg, followed immediately by alcuronium 15 mg. The patient rapidly became cyanosed and pulseless. The lungs were inflated with 100% oxygen via a facemask and the patient's trachea was intubated without difficulty. No laryngeal oedema was seen at laryngoscopy. Auscultation confirmed normal air entry in both lung fields and a Manley ventilator registered an airway pressure of 2.0 kPa. The cardiac monitor showed a heart rate of 40 beats/minute with a variable, irregular rhythm, which did not respond to atropine 0.6 mg. A bolus injection of isoprenaline 10 μg was given and 120 mmol of sodium bicarbonate infused. The patient's rhythm now changed to atrial fibrillation with a ventricular rate of 150 beats/minute and a palpable systolic blood pressure of 60 mmHg. The central venous pressure via an internal jugular catheter was 10 cm H_2O above the mid-axillary line (patient supine). Four DC shocks from 100–400 joules were required before stable sinus rhythm was achieved. Incremental doses of midazolam were given to maintain anaesthesia, to a total dose of 7 mg. At this time, serum potassium was 3.6 mmol/litre. Arterial blood gas analysis after the bicarbonate infusion, and with an $F\mathrm{IO}_2$ of 1.0, showed pH 7.453, $P\mathrm{CO}_2$ 4.14 kPa, $P\mathrm{O}_2$ 12.2 kPa, base excess 0.0 mmol/litre, bicarbonate 21.2 mmol/litre.

Surgery was deferred and the patient was transferred to the intensive care unit. Chest X ray and 12-lead electrocardiogram were normal. One and a half hours following the original arrest, a second episode of profound hypotension occurred. This time there was no dysrhythmia. Central venous pressure was now 6 cm H_2O. Five hundred millilitres of polygelin and 800 ml of plasma were rapidly infused, which restored normal peripheral perfusion after 40 minutes. Intermittent positive pressure ventilation (IPPV) of the lungs was continued overnight, with diamorphine for sedation. Cardiovascular stability now persisted, but there were two episodes of severe diarrhoea. The next day, before discontinuing IPPV, she underwent surgery with supplementary local anaesthesia. She made an uneventful recovery.

Further investigations

Electrocardiogram and cardiac enzymes at 24 and 48 hours after arrest were normal. Blood samples (50–100 ml) were collected and heparin added to give a final concentration of 50 units/ml. *In vitro* release of histamine from the patient's leucocytes was measured in response to incubation with various anaesthetic agents, including those used during her anaesthetic. This was done after the method described by Assem *et al.*[2] Intradermal skin testing was not performed due to patient noncompliance.

Results

Table 1 shows the release of histamine, expressed as a percentage of the total leucocyte content, induced by the agents tested. Thiopentone caused no release. Alcuronium produced a large release of histamine, highest at the 20 μg/ml concentration and less at 100 μg/ml, as would be expected if cross-bridging was essential for inducing histamine release. Minor cross-reactivity to vecuronium and fazadinium was seen, but not with the other five relaxants tested.

Table 1. Histamine release (% of total leucocyte content) caused by various agents tested.

Drug	Concentration of drug* (μg/ml)	Mean histamine release (%)
Control		0
Alcuronium	4	0
Alcuronium	20	17.2
Alcuronium	100	2.8
Atracurium	8–200	0
Suxamethonium	4–200	0
Tubocurarine	4–200	0
Pancuronium	4–100	0
Vecuronium	4	0
Vecuronium	20	1.0
Vecuronium	100	0
Thiopentone	4–100	0
Fazadinium	4	0
Fazadinium	20	1.6
Fazadinium	100	0
Gallamine	4–100	0
Pethidine	4–100	0

*Only the range of concentrations used is shown for those drugs with no histamine release.

Discussion

This reaction was presumably idiosyncrasy rather than allergy, since there was no previous exposure to alcuronium. Clinical observation alone may be entirely misleading in differentiating between anaphylactoid and allergoid reactions. The anaphylactoid reactions, mediated by histamine release, may exhibit some or none of the accepted criteria. End organ susceptibility is enormously variable. Allergoid responses, while less hazardous, normally exhibit cutaneous features only.[3] The patient's leucocytes reacted to alcuronium *in vitro* as shown by the abnormal histamine release. This substantially supports the diagnosis of anaphylactoid rather than allergoid reaction, specifically pointing to alcuronium as the cause.

The leucocyte tests are free of hazard to the patient as compared with *in vivo* challenge[4] and, to date, correlate well with clinical reactions. They are, however, expensive. Each comprehensive test takes a week and can involve an entire laboratory. The mechanism of histamine release from leucocytes *in vitro* is currently assumed to involve divalent binding of causative agent to leucocyte, which is somewhat similar to antigen-IgE antibody-mediated reactions (in true anaphylaxis).[5] Similarly, in the presence of drug or antigen excess, monovalent binding occurs and the extent of histamine release is reduced. This explains why a higher concentration than 20 μg/ml of alcuronium produced less histamine release.

Recent reports have supported the view that skin testing is unreliable in the management of suspected anaesthetic drug reactions.[6,7] However, prick tests, as opposed to intradermal tests, correspond well to the presence of allergen-specific IgE. Skin tests in general provide the only practical and relatively safe *in vivo* test available and should be used where possible to complete the identification of the causative agent. The significant incidence of false positive and negative reactions must be considered if they are the only investigation used. The use of a radio-allergosorbent test for drug allergy (RAST) which detects allergen-specific IgE, is not currently practical when applied to anaesthetic reactions, because of the difficulties in preparing hapten-carrier conjugates which are essential to this test. Measurement of total serum IgE levels is of limited value only. Raised levels would help to substantiate a diagnosis of drug allergy and suggest anaphylactoid (or even true anaphylactic) rather than allergoid reaction, but a low level does not exclude such a reaction.[8] Knowing the total serum IgE level does not help to identify the causative agent.

In this case, a diagnosis of anaphylactoid reaction was initially discarded because of the complete absence of bronchospasm and urticarial rash. The dramatic response to colloid during the second hypotensive episode suggested hypovolaemia for which there was no obvious cause. An atypical reaction was then reconsidered.

The first reported adverse reactions due to alcuronium consisted of episodes of moderate to severe hypotension.[9] Since then, greatest interest has been in Australia, where Fisher[10] reported an apparently high incidence of anaphylactoid reactions. Whether these were primarily cardiovascular, or also involved bronchospasm and skin rash, is unclear. Youngman *et al.*[11] noted that many reactions to muscle relaxants were purely cardiovascular. Chan and Yeung,[4] reporting two reactions to alcuronium in the same patient, noted an erythematous rash over the limbs and trunk prior to cardiovascular collapse. Both this patient and ours were noted to have severe diarrhoea following recovery. The mechanism of this is presumably histamine-mediated increase in gut motility and intraluminal fluid secretion.[3]

Acute circulatory collapse may be due to a fall in peripheral resistance as part of the histamine response. Relative hypovolaemia ensues. Low peripheral resistance seems unlikely as a cause of the initial collapse in this patient as the central venous pressure was 10 cm H_2O. However, most case reports of cardiovascular collapse following the use of muscle relaxants quote improvement of blood pressure and perfusion following rapid infusion of colloid or crystalloid. This currently seems to be the treatment of choice.

The heart has previously been suggested as a primary target organ in anaphylaxis.[12] Experimental anaphylaxis produced in animal heart preparations implicates histamine as a mediator of atrioventricular block, multifocal extrasystoles and sustained ventricular dysrhythmias.[12] In our case, bradydysrhythmia and myocardial dysfunction, without evidence of relative hypovolaemia, suggest this as the mechanism of collapse. As only a minute amount of isoprenaline had been given before the onset of atrial fibrillation, it seems probable that this dysrhythmia was due to continuation of the primary reaction. It is accepted that the positive chronotropic response to histamine is mediated by H_2 receptors.[3,12] This response can be eliminated in animal studies using H_2-receptor antagonists, such as cimetidine.[12] Atrioventricular conduction defects seem to be mediated by H_1 receptors and are selectively prevented by chlorpheniramine.[12] It would seem logical therefore to prevent the dysrhythmogenic and the latent negative inotropic effects of histamine (which are H_1-receptor mediated) with a combination of both types of histamine receptor antagonists. Human volunteers pretreated with H_1- and H_2-receptor antagonists do not elicit the anaphylactoid features sometimes associated with polygelin infusion.[3] However, the use of these antagonists to abort anaphylactic and anaphylactoid reactions in clinical practice is not universally accepted. The effects may not be dramatic, but these drugs should be considered on theoretical grounds alone. They may help to prevent continuation of the primary reaction and any further dysrhythmias. The exact role of the heart as a primary target organ in cardiovascular collapse associated with anaphylaxis has yet to be clarified.

Acknowledgment

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Anaesthesia, 1987, Volume 42, pages 168–170

CASE REPORT

Respiratory depression after intrathecal opioids

Report of a patient receiving long-term epidural opioid therapy

T. KRANTZ AND C. B. CHRISTENSEN

Summary

Morphine 20 mg and pethidine 50 mg were accidentally injected intrathecally in a patient who had received large doses of opioids epidurally for cancer pain and who had shown tolerance to their effects. The well established tolerance to spinal opioids did not protect the patient against a moderate degree of respiratory depression. Morphine concentrations 6.5 hours after the morphine injection were 103 500 ng/ml and 52 ng/ml in cerebrospinal fluid and serum, respectively.

Key words

Anaesthetic techniques, regional; spinal, epidural.
Complications; respiratory depression.

The effectiveness of spinal opioid therapy for alleviation of cancer pain is well recognised, but the development of tolerance to the opioid is a serious limitation of such therapy.[1] We present here a case of accidental intrathecal injection of morphine and pethidine, possibly due to catheter tip displacement during high-dose epidural morphine therapy for cancer pain.

Case history

A 60-year-old male was hospitalised for carcinoma of the prostate with metastases in the lower lumbar vertebrae, pelvic bones and left cerebellar hemisphere. He complained of low back pain radiating to the lower extremities and, after oral opioid therapy had failed, a lumbar, epidural catheter was inserted percutaneously and long-term epidural morphine therapy was initiated. Over a period of 5 weeks the epidural morphine dose was increased from 4 mg in 10 ml saline three times a day to 10 mg in 10 ml saline six times a day. This therapy was continued for 19 weeks on an outpatient basis with good pain relief.

The patient was then rehospitalised and, over a period of 7 weeks, the dose was increased to 20 mg in 10 ml saline seven times a day and finally supplemented with 50 mg pethidine in 10 ml saline. This did not produce any pain relief or side effects. Adjunctive daily oral therapy was with paracetamol 3 g, diazepam 10 mg, levopromazine 25 mg, prednisone 30 mg and diuretics. Furthermore, 10 mg of ketobemidone was administered intramuscularly 12 hours before the last epidural morphine injection.

The catheter had been re-inserted 2 weeks earlier and tested with 3 ml of mepivacaine 2% without evidence of spinal anaesthesia. It was therefore felt that subcutaneous displacement of

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the catheter might be responsible for the therapeutic failure. A test dose of 3 ml 2% mepivacaine was injected 4 hours after the last morphine injection. Within 5 minutes this produced anaesthesia to T_8 with hypotension and lower limb paralysis. The patient became pain free, but was drowsy and was transferred to the intensive care unit where he was nursed in the supine position. A lumbar puncture was performed 6.5 hours after the last morphine injection and samples of cerebrospinal fluid (CSF) and serum were sent for morphine analysis by a specific radioimmunoassay.[2] Morphine concentrations were 103 500 ng/ml and 52 ng/ml in CSF and serum, respectively.

The patient was still pain free after the spinal anaesthesia wore off but his respiratory rate decreased transiently to 7 breaths/minute at 8, 12 and 13 hours after the last morphine injection, although it could be raised by verbal command. Naloxone 0.04 mg was injected intravenously 13 hours after the last morphine injection and the respiratory rate increased immediately to 12 breaths/minute; the patient became more alert and complained of slight pain, which ~~responded to~~ ketobemidone chloride 2.5 mg given subcutaneously.

The respiratory rate remained at 8 breaths/minute or above during the rest of the stay in the intensive care unit; it was 13 breaths/minutes at 22 hours after the last morphine injection, when the patient was pain free and considered to be in his usual condition. In the ward, oral morphine therapy was started with a slow-release preparation, but after 9 days the need for analgesics rapidly increased and finally necessitated intravenous opioid therapy, until he died 3 weeks later.

Discussion

To the authors' knowledge respiratory depression due to epidural[1] or intrathecal[3–13] administration of opioids in cancer patients with well established tolerance to opioids has not been reported previously, although epidural morphine infusion of 10 mg/hour,[14] single intrathecal injections of 20 mg[15] and intrathecal morphine infusion of 75 mg/day have been used.[16]

In our patient, the occurrence of spinal anaesthesia and the high morphine concentration in the CSF 6·5 hours after the last morphine injection strongly suggest that 20 mg of morphine and 50 mg of pethidine were injected intrathecally. Since this did not render the patient pain free, tolerance to the effects of spinal opioids must have occurred; further therapy of this type would therefore have been futile. The sedation can be explained by the patient becoming pain free after the intrathecal mepivacaine. The clinically moderate respiratory depression evident 8 hours after the last morphine injection is in accordance with the observations of others, although the onset can be quite variable and quicker when pethidine is used.[1,3] The dose of naloxone used by us to counteract the respiratory depression, was less than reported by others[5,7,9–12] and the slight analgesic reversal by naloxone contradicts recent reports.[5,6,9]

The apparent beneficial effects of the small dose of opioid administered subcutaneously might suggest psychological factors. The opioid tolerance was reversed to some degree by the spinal anaesthesia, as the duration of freedom from pain outlasted the sensory motor blockade. The reversal of tolerance to spinal opioids by epidural anaesthesia has been reported before[17] and has been attributed to a pain-alleviating mechanism due to blockade of sympathetic nerves.[18]

Taking the kinetics of a single, low-dose intrathecal morphine injection as a rough guide, and assuming an apparent CSF distribution volume of 70 ml and a one-compartment model with a half-life of 3.2 hours,[19] a calculated CSF morphine concentration 6.4 hours after injection of 20 mg intrathecally would be approximately 71 500 ng/ml. After intrathecal morphine injection of 0.2 mg/kg, plasma values of morphine 6.5 hours after injection have been shown to be about 10–20 ng/ml.[20] Both values are somewhat lower but in broad accord with our values.

Finally, we wonder whether the catheter tip migration through meningeal converings in the course of 2 weeks might have been facilitated by the adjuvant glucocorticoid therapy, which has a well-known inhibitory effect on connective tissue repair.[21]

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Anaesthesia, 1987, Volume 42, pages 171–174

CASE REPORT

Unilateral pulmonary oedema/atelectasis in the lateral decubitus position

A. BARAKA, R. MOGHRABI AND A. YAZIGI

Summary

An obese male patient developed hypoxia, hyercarbia and radiological signs of pulmonary oedema/atelectasis in the dependent lung after surgery in the lateral decubitus position. This appears to have been due to ventilation–perfusion mismatch, although other factors were considered. The patient recovered following 36 hours of intermittent positive pressure ventilation of the lungs.

Key words

Anaesthesia; urological.
Complications; atelectasis, pulmonary oedema.

The dependent lung receives relatively more perfusion than the nondependent lung,[1] in the anaesthetised patient positioned in the lateral decubitus position. However, the majority of tidal ventilation is switched to the nondependent lung,[2-4] which results in mismatching of ventilation and perfusion. The present case report shows that unilateral pulmonary oedema and atelectasis can also occur as complications of the lateral decubitus position.

Case history

The patient was a 50-year-old male, who weighed 100 kg. He suffered from cancer of the right kidney and was scheduled for an elective radical nephrectomy. Pre-operatively, the patient was moderately hypertensive (blood pressure was 160/95 mmHg); examination of the chest and heart showed no abnormality. The patient was premedicated by intramuscular injections of morphine 10 mg and scopolamine 0.3 mg. Anaesthesia was induced with thiopentone 5 mg/kg and suxamethonium 1.5 mg/kg. Orotracheal intubation was easily performed, using a cuffed tube size 9.0 mm. Chest auscultation revealed bilaterally equal breath sounds. Anaesthesia was maintained with nitrous oxide and oxygen (2:1 litres/minute) using a circle system with carbon dioxide absorption. Neuromuscular blockade was maintained with alcuronium. Ventilation was mechanically controlled throughout the surgical procedure (Ohio Anaesthesia Ventilator) at a rate of 15 breaths/minute. The patient was positioned in the kidney position with the right side uppermost. Auscultation of the chest after positioning the patient confirmed the appropriate position of the tracheal tube. Arterial blood gas analysis after 30 minutes showed oxygen tension (Pa_{O_2}) 18.5 kPa, carbon dioxide tension (Pa_{CO_2}) 4.3 kPa and pH 7.42. The surgical procedure was associated with

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an estimated blood loss of about 10 units, which necessitated the transfusion of 4 units of rewarmed whole blood, 4 units of fresh frozen plasma and 4 litres of lactated Ringer's solution. The blood pressure was maintained near its control value and the central venous pressure (CVP) at 5–10 cm H_2O. Near the end of surgery, which lasted 4.5 hours, the blood gas analysis was repeated with the same ventilation and inspired oxygen concentration, and showed a reduction of Pao_2 to 9.3 kPa, elevation of $Paco_2$ to 5.7 kPa, and decreased pH to 7.32. Chest auscultation confirmed bilateral ventilation of both lungs; breath sounds were slightly less in the dependent lung.

Neuromuscular blockade was antagonised at the end of surgery by a mixture of neostigmine 0.05 mg/kg and atropine 0.02 mg/kg. Blood gas analysis after tracheal extubation, while the patient was spontaneously breathing oxygen delivered by a facemask, showed Pao_2 6.7 kPa, $Paco_2$ 5.7 kPa and pH 7.32. A chest X ray revealed unilateral pulmonary oedema and possible atelectasis of the left lung (Fig. 1). The trachea was re-intubated and intermittent positive pressure ventilation was started with Fio_2 0.5, respiratory rate 10 breaths/minute, tidal volume 1100 ml and positive end expiratory pressure (PEEP) 0.5 kPa. Arterial blood gases improved: Pao_2 13.1 kPa, $Paco_2$ 5.1 kPa and pH 7.36. After 12 hours of controlled ventilation and physiotherapy, the chest X ray showed marked aeration of the left lung (Fig. 2). Arterial blood gas analysis also showed further improvement: Pao_2 16 kPa, $Paco_2$ 4.1 kPa and pH 7.43. It was possible after 36 hours to allow the patient to breathe spontaneously.

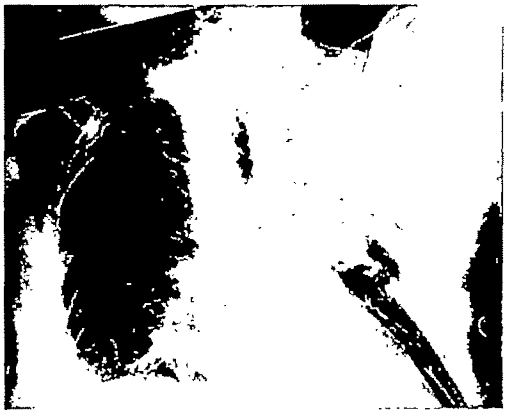

Fig. 1. Chest X ray taken in the immediate postoperative period to show pulmonary oedema and possible atelectasis of the left lung.

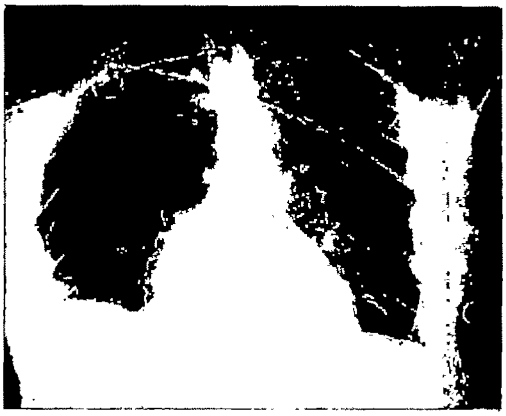

Fig 2. Chest X ray taken 12 hours postoperatively to show marked aeration of the left lung.

Discussion

This case report describes the occurrence of unilateral pulmonary oedema and/or atelectasis of the dependent lung in an obese patient who was operated on in the lateral decubitus position. This complication may be attributable to accidental intubation of the right main bronchus; however, repeated chest auscultation following tracheal intubation and after positioning, revealed bilateral and equal breath sounds in both lungs. Arterial blood gas analysis 30 minutes after positioning showed normal Pao_2 and $Paco_2$ levels as further evidence for two-lung ventilation. The development of unilateral pulmonary oedema may have been secondary to overtransfusion. However, fluid replacement in our patient matched the estimated blood loss and was never associated with elevation of the CVP above 5–10 cm H_2O. Although we cannot definitely exclude unilateral bronchial intubation, overtransfusion or pulmonary aspiration as possible causes of unilateral pulmonary oedema and/or atelectasis in our patient, the prolonged period in the lateral decubitus position *per se* can predispose to this complication.[5]

In the awake patient positioned in the lateral decubitus position, the dependent lung receives relatively more perfusion and ventilation than the nondependent lung;[6] thus, the ventilation–perfusion ratio of each lung remains approximately constant, regardless of position. During anaesthesia, the dependent lung continues to receive more perfusion than the upper lung, but the majority of ventilation is switched to the nondependent lung;[2–4] this results in a mismatch of ventilation and perfusion. The nondependent lung is well ventilated but poorly perfused, which results in a dead-space effect, while the dependent lung is more perfused than ventilated and results in a shunt-like effect.[1] Such ventilation–perfusion mismatch can explain the reduction of $P\mathrm{aO_2}$ and elevation of $P\mathrm{aCO_2}$ which developed in our patient.

The functional residual capacity (FRC) of both lungs is decreased on induction of general anaesthesia.[7] Since each lung occupies a different initial position on the pulmonary pressure–volume curve while the patient is awake, anaesthesia-induced reduction in each lung's FRC causes the nondependent lung to be more compliant, while the dependent lung becomes less compliant.[8] The decreased compliance of the dependent lung is exaggerated by physical impedance of the lower lung expansion by the weight of the mediastinum and the abdominal contents, as well as by the pressure of the supporting rolls and operating table angulation. All these factors, in addition to possible accumulation of secretions in the dependent airways, can result in inadequate ventilation and even atelectasis of the dependent lung.[5]

In the lateral position, there is also an increased perfusion of the dependent lung. The pulmonary circulation, being a low-pressure system, remains quite gravity-dependent. The majority of the pulmonary blood flow goes to the dependent lung,[1] which results in increased pulmonary capillary pressure in the dependent lung, with a consequent increase of fluid transudation. Thus, the increased gravitational perfusion of the dependent lung, combined with its low FRC and decreased ventilation, can cause the dependent lung to be at increased risk of unilateral pulmonary atelectasis and/or oedema.[5] In our patient, the problem might have been magnified by the prolonged operative time, excessive crystalloid infusion and by obesity, which can produce further reduction of FRC and increase of the closing volume.[9]

The application of positive end expiratory pressure (PEEP) during surgery in the lateral decubitus position might be used to restore the majority of ventilation to the lower lung, presumably by moving the lower lung up to a steeper and more favourable part of the pressure–volume curve and the upper lung to its original position in a flat, unfavourable portion of the curve.[8] A more physiological solution to this ventilation–perfusion mismatch in the lateral decubitus position would be differential ventilation with the application of selective PEEP to the dependent lung.[11,12] Selective PEEP increases the FRC of the lower lung and counteracts the gravitational increase of dependent lung perfusion, leading to more even matching of ventilation and perfusion.

In conclusion, ventilation is preferentially distributed to the nondependent lung in the lateral decubitus position in anaesthetised patients, whereas perfusion is distributed mainly to the dependent lung. This results in ventilation–perfusion mismatch which can cause hypoxaemia and carbon dioxide retention. The decreased FRC of the dependent lung, associated with its gravitationally increased perfusion, may also be complicated by unilateral pulmonary atelectasis and/or oedema.

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APPARATUS

Oxygen analysers

An evaluation of five fuel cell models

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Summary

Five currently available fuel cell oxygen analysers were studied with a view to their use in anaesthesia. The accuracy, response time and safety features of these analysers are discussed. Fuel cell analysers appear to be suitable oxygen monitors for routine anaesthetic use.

Key words

Equipment; oxygen analysers.

Monitoring of the inspired oxygen concentrations during anaesthesia is becoming increasingly widespread despite the use of calibrated rotameters. The rising incidence of litigation has resulted in recommendations that the use of analysers should be mandatory, to avoid the administration of an hypoxic mixture to any patient.[1–3] The introduction of new and more expensive volatile anaesthetic agents has caused renewed interest in the use of low-flow breathing systems in an effort to reduce expenditure. In these systems it is essential for inspired oxygen concentrations to be measured, since the machine rotameters provide no indication of the gaseous composition of inspired mixtures.

An oxygen analyser used for anaesthesia must be accurate, despite the presence of other gases and water vapour. Polarographic cells are influenced by the presence of nitrous oxide in the gas mixture.[4,5] The new generation of oxygen analysers utilise C_2 galvanic cells which may be used in the presence of nitrous oxide. In this study we have examined the performance of five galvanic cells of the C_2 type, under conditions likely to be found in anaesthetic breathing systems.

Description of monitors

The following analysers were studied: Miniox III (Catalyst Research Corporation); Oxydig (Draeger); Model 251 (Vickers Medical); MUA 210 (Vial Medical); Ohio 5100 (Ohmeda). These monitors all use a C_2 type fuel cell, which is a modification of the earlier C_1 type cell which could not be used in the presence of nitrous oxide. The cells consist of two electrodes: a gold cathode, exposed to the gas through a fluoropolymer membrane, and a lead anode submerged in potassium hydroxide. Oxygen diffusing through the membrane is electrochemically reduced at the cathode and a corresponding oxidation occurs at the anode:

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$$\begin{array}{ll}\text{Cathode} & O_2 + 4e \rightarrow 2\,O^{2-} \\ \text{Anode} & 2\,O^{2-} \rightarrow O_2 + 4e\end{array}$$

The current generated is proportional to the partial pressure of oxygen. No external power source is required by the cell.[6]

The Miniox III, as originally supplied in North America, has a short sensing arm which fits into the requisite T-piece used there. However, the British suppliers use an alternative T-piece and have produced a modified sensing arm. This was used in the tests. Draeger have recently introduced a modified version of the Oxydig. The performance specifications are similar, but certain design and operational characteristics have been altered. Tests were carried out on the original, but the new model has been inspected and some preliminary tests have been carried out.

The Ohio 5100 sensor possesses two cells. The monitor has a switch to turn to the alternative cell in the event of failure of the first cell. The Oxydig (Draeger) also possesses two cells. The monitor displays a mean of both cell readings. If these readings disagree by more than a given tolerance, the monitor enters an inoperative mode.

All the monitors except the Model 251 (Vickers) use batteries to power the display circuitry and alarms. The Model 251 simply measures the current produced by the fuel cell and displays it on an analogue screen with no alarms. Table 1 lists certain features of the monitors under study, according to the manufacturers' published specifications. The figure for 5-year cost was calculated by adding the price of the monitor to 5 years' supply of cells, using the manufacturer's estimate of cell life in normal use. Prices were accurate for February 1986.

Method

The monitors were calibrated according to manufacturers' specifications, at ambient temperature and pressure. They were then incorporated into the circuit illustrated in Fig. 1. Steady state readings at varying oxygen concentrations from 0 to 100% and then back to 0%, using between five and ten points in each direction, were taken. The monitors were allowed to settle at a steady state reading for at least 5 minutes at each concentration. The analysers were studied using dry gas at ambient temperature and pressure, with oxygen diluted with nitrogen, nitrous

Table 1. Table of costs and other features of monitors according to manufacturers' specifications.

	Miniox III	MUA 210	Vickers	Ohmeda	Draeger
Monitor cost (£)	695	420	225	620	580
Sensor cost (£)	75	75	68	115	103
Sensor life (months)	12	8	3	12	12
Five-year cost (£)	1070	983	1585	1195	1095
Calibration point	100%	21%	21%	100%	100% & 21%
Calibration method	Automatic	Manual	Manual	Manual	Automatic
Alarms	Hi/Lo	Hi/Lo	Nil	Hi/Lo	Hi/Lo
Alarm limits	<18%, >99%	Unspecified	—	<18%	<18%
Display	Dig. LED	Dig. LED	Analogue	Dig. LED	Dig. LED
Display resolution as % of full scale deflection	0.1	1	2	1	1
Power	Battery	Battery	—	Battery	Battery

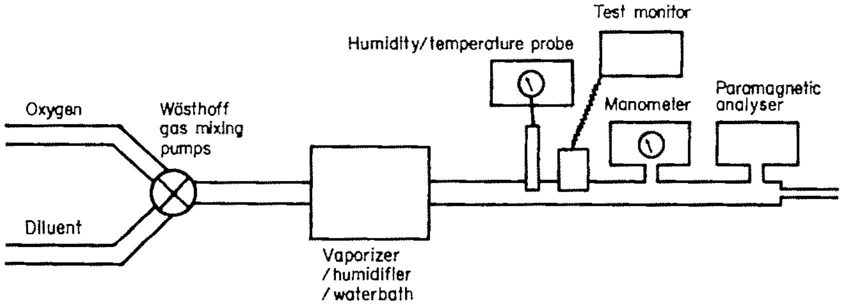
Test monitor
Humidity/temperature probe
Paramagnetic analyser
Oxygen
Wösthoff gas mixing pumps
Manometer
Diluent
Vaporizer /humidifier /waterbath

Fig. 1. Diagram of circuit used.

oxide and nitrous oxide plus 2% halothane. The studies with nitrous oxide were repeated at 37°C with both dry gas and at 100% relative humidity, and at ambient temperature at 30 cm water pressure (2.94 kPa). These cover a range of conditions one might expect to encounter in anaesthetic and intensive care work.

Gas mixtures were provided using gas mixing pumps (Wõsthoff) with a nominal gas flow of 850 ml/minute, except in the increased temperature experiments when 4 litres/minute from flowmeters calibrated against an Ohio spirometer were used. Accurate assessment of oxygen concentrations was obtained using a Servomex paramagnetic oxygen analyser, accurate to within 0.1% oxygen according to the manufacturer's specification, which had been calibrated at three points using oxygen, nitrogen and air. Gas pressure in the system was monitored continuously with an aneroid gauge (Blease Anaesthetic Equipment) calibrated against a water manometer. Oxygen and nitrous oxide were obtained from hospital pipelines. Nitrogen and air were obtained from cylinders. The temperature of the gas was measured using a needle probe thermocouple and displayed on an Electrolaboratoret mirror galvanometer. Halothane was added by incorporating into the circuit a Fluotec Mark 2 vaporizer (Cyprane) calibrated against an anaesthetic vapour monitor (Datex) for the fresh gas flows used. A temperature of 37°C was obtained using a warming coil in the water bath. Higher gas flows in the circuit were found to be necessary in order to maintain the raised temperature at steady state. Humidification was obtained by incorporating a cascade humidifier (Bennett) in the circuit. Humidity was measured using a humidity probe (Vaisala HM114). Atmospheric pressure and ambient temperature were recorded during each experiment.

The paramagnetic analyser was not used in the experiment carried out with raised humidity as readings would be affected by water vapour. The settings of the Wõsthoff pumps were therefore taken and a correction made for the effects of 100% relative humidity on the oxygen partial pressure. Increased pressure (3.0 kPa) was produced by applying a restriction to the tubing distal to the sensor. The pressure was monitored continuously using the aneroid gauge. The paramagnetic analyser was not subjected to this high pressure and was situated distal to the constriction. A correction was made for the effects of increased pressure on oxygen partial pressure.

Response times were measured for each monitor from 0–21% oxygen and 100–21% oxygen. The sensor was used with its own T-piece in circuit, at a flow of 5 litres/minute at ambient pressure. A three-way tap was used to change gases and was situated close to the sensor (estimated delay > 1 second). The reading of the monitor was noted at 5-second intervals until a steady level had been attained. A curve was plotted and the time taken to reach 90% of the final value was estimated. All runs were carried out at the same ambient temperature and pressure.

Results

For each monitor, under each set of conditions, results were plotted on a scatter diagram of test monitor reading against standard reading (Servomex), to look for any trend in the error. Two representative scatter diagrams are shown in Figs 2 and 3. The Oxydig (Draeger) shows a linear response, whereas the MUA 210 (Vial Medical) shows some error due to hysteresis.

Error was calculated by taking the difference between the test monitor reading and the Servomex reading. Mean error was calculated for all the readings under each condition and represents the deviation from the ideal line $y = x$. A 95% confidence interval for each condition was also calculated. The error should lie between these limits, either side of $y = x$ in 95% of cases. Mean error and 95% confidence intervals, expressed in terms of percentage of full scale reading (100% O_2), are given to one decimal place in Table 2. Ninety-percent response times are shown in Table 3.

Discussion

An oxygen monitor suitable for use in anaesthesia must be accurate, to a degree necessary for clinical decisions to be made. It must not be affected by the various physical conditions encountered in practice. It must have a rapid response time with effective alarms to alert the attendant to dangerous oxygen concentrations and it must be stable and reliable over prolonged periods of use. Its use must not be associated with any increased danger to the patient and it must be easy to set up to use and be of reasonable cost.

All monitors studied were sufficiently accurate

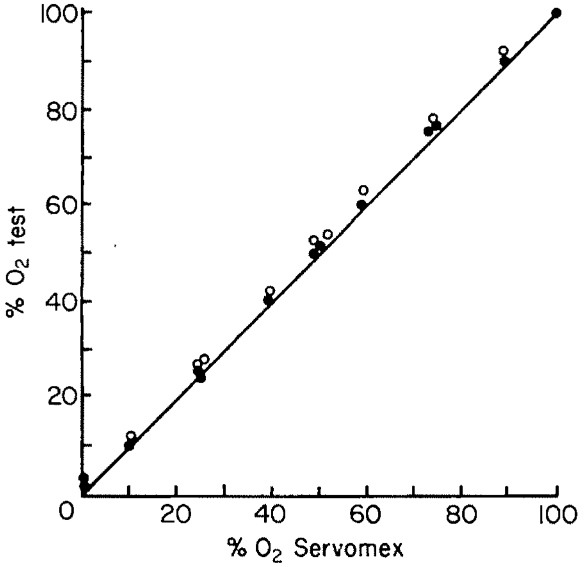

Fig. 2. Graph of test reading on MUA 210 (Vial Medical) versus reading on Servomex. Mean error 1.5%; 95% confidence interval, ± 1.88; straight line shows $y = x$; line of best fit is $y = 4.6 + 0.9x$. ●, Upward run; ○, downward run.

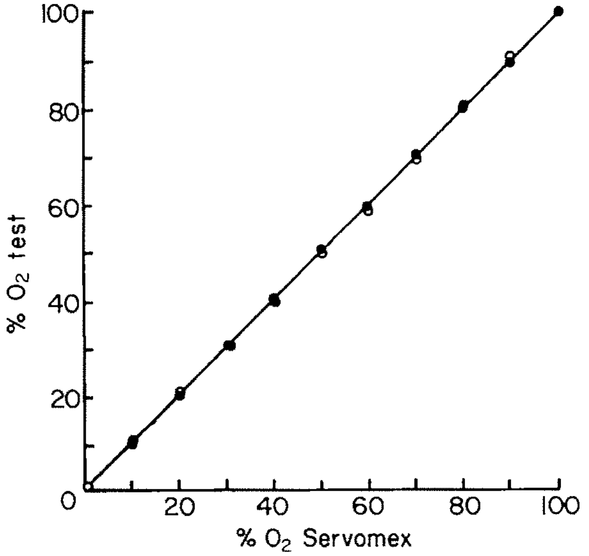

Fig. 3. Graph of test reading on Oxydig (Draeger) versus reading on Servomex. Mean error 0.7%; 95% confidence interval, ±0.96; straight line shows $y = x$; line of best fit is $y = -0.56 + x$. ●, Upward run; ○, downward run.

Table 2. Performance at steady state. Mean error with 95% confidence interval expressed as a percentage of full scale deflection. These results have been corrected to allow for effects of pressure and humidity on the partial pressure of oxygen.

	Miniox III	MUA 210	Vickers	Ohmeda	Draeger
N_2/O_2	0.5 (0.47)	1.5 (1.88)	0.8 (1.76)	0.7 (1.10)	0.7 (0.96)
N_2O/O_2	1.0 (0.96)	1.4 (2.39)	0.8 (1.25)	0.8 (1.00)	0.9 (1.39)
N_2O/O_2/halothane	0.6 (0.82)	2.1 (3.61)	1.0 (1.49)	0.5 (0.67)	0.6 (0.59)
N_2O/O_2/37°C	1.0 (1.02)	3.0 (4.39)	2.2 (4.06)	1.2 (1.61)	0.7 (0.55)
N_2O/O_2/100% RH/37°C	0.7 (0.69)	6.5 (8.43)	0.8 (1.08)	0.5 (1.06)	0.8 (1.23)
N_2O/O_2/2.94 kPa	0.5 (0.74)	1.7 (2.59)	0.84 (1.39)	0.5 (0.92)	1.4 (2.21)
Spec. accuracy in N_2	1.0	—	2.0	1.0	3.0

Table 3. Table of 90% response times in seconds.

	Miniox III	MUA 210	Vickers	Ohmeda	Draeger
0–21%	11	32	26	13	13
100–21%	9	48	42	12	12
Specified response time	12	—	30	15	20

for clinical purposes. The mean error on the MUA 210 for oxygen in nitrogen of 1.5% was predominantly due to overreading. Hysteresis was noted and the overreading was more marked on the downward run. The maximum error was 3.8%. Despite a built-in temperature compensation device, the overreading was more pronounced at 37°C (mean error 3.0%) and with 100% relative humidity (mean error 6.5%). A metal grille between gas and sensor on the MUA 210 became heavily coated with condensation in the humidity experiment and could account for the error. Overreading also occurred at 3.0 kPa pressure in this monitor, but the hysteresis was less marked. The other monitors were not affected to a notable extent by the applied conditions. None of these C_2 cells showed an increased error of greater than 0.5% in nitrous oxide when compared to its performance in nitrogen, although the 95% confidence limits on the MUA 210 increased from ±1.88 to ±2.39. Halothane was similarly without effect on the readings of all monitors except the MUA 210 whose mean error increased from 1.5 to 2.1% with a widening of 95% confidence limits from ±1.88 to ±3.61, showing an increased scatter of readings. Response times were adequate on all models, but the extra delay on the MUA 210 and the Vickers could lose vital seconds in an hypoxic situation. Pilot studies using the MUA 210 showed that response times were improved at higher pressures. This probably explains why there was little hysteresis at raised pressure. The manufacturers have recently altered their T-piece to an L shape to place the sensor mouth directly in the gas stream. Response times do not appear to have been affected, however, according to the preliminary tests which we have carried out on this new T-piece.

The Miniox III and the Draeger Oxydig both have sensor mounts (fins and angled T-piece, respectively) designed to produce turbulence, and both have very rapid response times. The response times on the Ohmeda were within specifications when measured at 25°C as recommended by the manufacturer. They were, however, prolonged to >30 seconds when measured at 17°C. This was not noted on the other monitors.

The presence of a T-piece in an oxygen sensor increases the risk of leakage and disconnexion in a breathing system, for example in the inspiratory limb of a Manley ventilator system, where any leakage will reduce the minute volume and the inspiratory pressure. Manufacturers' T-pieces fit with standard equipment, but it is advisable to check for leaks in each situation as individual connexions may vary. No sampling gas is removed from the system and the oxygen consumption by the cell is minimal.

None of the manufacturers gives any indications of electrical safety of the equipment and we carried out no tests on this. The Draeger Oxydig is specified to be within ISO and IEC requirements. The sensors of both fuel cell and polarographic analysers sit in the gas stream and are thus subject to more stringent safety regulations. Restriction of supply voltages is recommended for polarographic cells. Fuel cells, however, have no need for external power supply and are thus free from this problem. No information is provided by manufacturers with respect to the involvement of sensors and sensor mounts in leakage currents.

All monitors were small and portable and easy to use. All displays were adequate in good lighting. The Draeger provides illumination of the display if required. All controls were found adequate, except the original Draeger, whose rubber buttons were poorly labelled, hard to depress and gave little tactile sense of having responded. These problems have, however, been corrected on the new model. The Vickers 251 possesses no alarms and is thus at a disadvantage against its competitors. All other alarm systems were adequate, but the lack of a low setting limit on the MUA 210 gave cause for concern, as it could not be trusted to be active in an hypoxic situation. The manufacturers are working on a change of internal programming to rectify this. All alarms are set to integer percentages and are accurate to this, with an intrinsic error of ±0.5%. This does not add an appreciable error to the oxygen concentration at which the alarm is activated.

Calibration of the monitors was straightforward on all models. Those monitors with manual calibration required adjustment of a gain control until the correct reading was obtained for a standard O_2 concentration. Those with automatic calibration simply required a button to be pressed in the presence of the standard O_2 concentration. This puts the monitor into calibration mode and it returns to normal reading automatically when calibration has been effected. This takes a maximum of 30 seconds. The original Draeger Oxydig possessed an automatic calibration request which was activated whenever recalibration was considered necessary. We encountered one problem with this as it was automatically activated at readings $>100\%$. Often, following calibration at 100%, the monitor would read over 100% and go into the 'calibration request' mode repeatedly. This has been overcome in the new model, which reads up to 105%, allowing for the ±3% accuracy claimed by the manufacturers. The new Draeger Oxydig, however, enters an 'inoperative' mode at oxygen concentrations of less than 5% and will not read any further until switched off/on and recalibrated at two points. Thus, in an hypoxic situation the attendant is left without a guide as to whether his restorative measures have been effective in increasing the inspired oxygen, which may not be the case if a pipeline cross-over has occurred.[1,7–9]

The inclusion of many safety cut-out features has led some anaesthetists to argue against the routine use of oxygen analysers. Multiple false alarms have led to analysers being switched off.[10] Others, however, have contended that with careful use, such analysers are practical.[3] Recent galvanic oxygen analysers, as tested here, appear to be rapid and accurate enough to act as a suitable oxygen monitoring device during anaesthesia. We feel that the inclusion of mandatory alarms for low oxygen concentrations ($<18\%$) is essential. We believe that a well chosen and correctly used oxygen analyser would lead to increased safety during anaesthesia but is, of course, no substitute for continual vigilance on the part of the attendant.

Acknowledgments

We would like to express our appreciation of the help of the late Professor C.M. Conway of the Magill Department of Anaesthetics, and to thank Dr T.R. Williams and Mr D.A.P. Strickland of the Department of Clinical Measurement at Westminster Hospital for helpful discussion in the preparation of this paper. We would like to thank the Department of Medical Illustration, Westminster Hospital for help in the preparation of the figures and Mrs S. Braham for typing the manuscript. We would also like to thank the respective manufacturers for loan of the oxygen analysers.

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Anaesthesia, 1987, Volume 42, pages 182–185

APPARATUS

The severance of epidural catheters

G. L. HUTCHISON

Summary

Four groups of Portex Minipack epidural sets were tested to measure the force required to sever the catheter on the needle bevel. The 18-gauge radio-opaque catheter was found to sever at a significantly lower applied force and to stretch significantly less before breaking than the 18-gauge clear catheter or either of the 16-gauge catheters. The other catheters tested had breaking strains of between 10–20 Newtons (1–2 kg force) and stretched by more than 30% of their lengths before breaking. It is suggested that these other catheters may therefore be a preferable alternative to the 18-gauge radio-opaque.

Key words

Equipment; catheters, epidural.

Reports of problems with the use of epidural catheters include misplacement,[1,2] occlusion,[3] faulty manufacture,[4,5] knotting[6] and difficulty with removal.[7] Despite mention in textbooks[8] and product information, the complete division of a catheter by withdrawing it through the needle is rarely reported. On these occasions a faulty needle may be involved.[6] More commonly, a catheter breaks when being removed from the patient at the end of its use. Partial division on the needle bevel at the time of insertion has been implicated,[9] although nipping of the catheter by the spinous processes may also be a cause.[10]

Dawkins[11] reported a 0.1% incidence of catheter breakage in caudal anaesthesia, and Bonica *et al.*[12] had a similar frequency of breakage with epidural blocks (three in 3637). In 1985, Crawford[13] reported 12 broken catheters out of 27 057 epidurals over a 17-year period and noted that none of these cases had occurred in the last 5 years, since the adoption of modern Portex epidural catheters in his centre. This observation, together with Dawkins' mention in 1969 of a catheter being broken by 'the hand of a porter lifting the patient . . .,'[11] suggests that present-day epidural catheters are more resistant to damage than their older counterparts.

The objective of this investigation, therefore, was to establish the force required to damage a modern epidural catheter on the needle.

Methods

Radiographic studies[12,13] have shown that epidural catheters frequently curl up at the site of entry to the epidural space. It is theoretically possible, therefore, for a catheter to kink around the needle bevel in such a way that it will not easily withdraw through the needle again, but will tend to lock in place. This situation would predispose the catheter to damage if attempts at withdrawal through the needle were made. It was, however, very difficult to find a reproducible method of so kinking the catheter for

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experimental purposes. Even when bent at an angle of 30–45° to the shaft of the needle, catheters would frequently slip easily back through the needle with gentle traction.

Because of this difficulty in simulating the clinical conditions, the apparatus illustrated (Fig. 1) was used. The catheter hub and the needle were firmly fixed by clamps and the catheter was led at a 45° angle from the hub, through the needle (which was held vertically) and attached to a receptacle of negligible weight (a plastic bag weighing 2–3 g). Weights, from a standard balance-type weighing machine, were added to this receptacle in increments of 100 or 200 g, so as to apply increasing traction on the catheter. Sixty seconds were allowed to elapse between the addition of each weight increment, during which time the catheters stretched to a new equilibrium length.

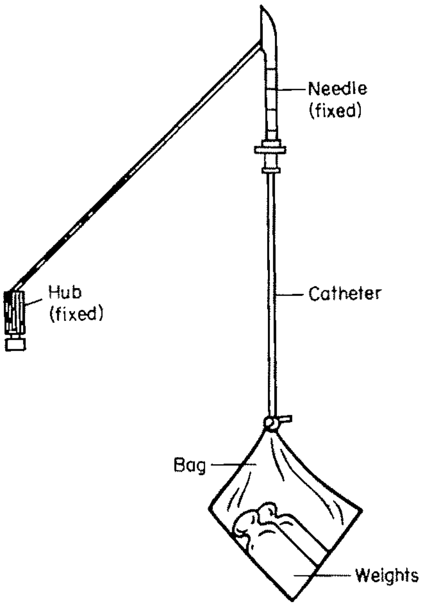

Fig. 1. Equipment used. Hub and needle held rigidly by clamps in the relationship illustrated.

At the start of each test, and just before the addition of a new weight increment, the length of the catheter was measured from the point of suspension as it crossed the needle bevel to the point of attachment to the weights. The proportional increase in length of each catheter was then calculated for each weight increment. This process was repeated until the catheter broke.

Table 1. Catheter/needle combinations tested.

Portex 18-gauge Minipack—radio-opaque catheter
Portex 18-gauge Minipack—clear catheter
Portex 16-gauge Minipack—radio-opaque catheter
Portex 16-gauge Minipack—clear catheter

Four groups of catheters and needles were tested, from the Portex Minipack range (Table 1). Seven tests were made in each group and a new catheter and needle were used for each test. Results were analysed in terms of the weight required to break each catheter, the maximum increase in length measured before the catheter broke ('stretch before breaking') and the graphs of stretch against applied weight for each type of catheter. Student's *t*-test and the Wilcoxon rank sum test were used for statistical analysis.

Results

All the catheters broke at the point of suspension, that is, where they crossed the chisel edge of the needle bevel. Several catheters took up to 30 seconds to break after an increase in weight had been applied.

Breaking strains

The weights at which the catheters were severed are illustrated in Fig. 2. As might be expected, the 16-gauge catheters supported significantly more weight than their 18-gauge equivalents ($p < 0.005$). There was no difference in breaking strain between the two 16-gauge catheters, but the 18-gauge radio-opaque catheter divided significantly more easily than the 18-gauge clear catheter ($p < 0.05$).

Stretch curves

For an elastic material, Hooke's Law states that increase in length is proportional to applied force, at forces below the elastic limit of the material. When the elastic limit is exceeded, stretch increases rapidly with further increases in force. These two phases are most clearly seen in the curve for the 18-gauge radio-opaque catheter (Fig. 3). Most catheter breakages occurred in the second, inelastic phase of stretching. The curves for the two 18-gauge catheters (Fig. 3) are significantly different ($p < 0.05$) at all values above 400 g; the clear catheter stretched more in response to a given applied weight than the radio-opaque catheter.

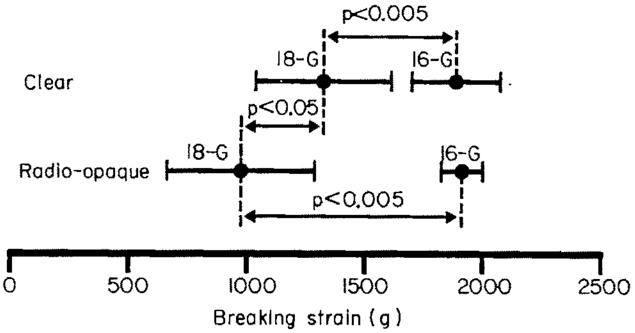

Fig. 2. Breaking strains. Means (SD) (⊢●⊣). Significance values relate to the difference between the groups bracketed (Student's *t*-test).

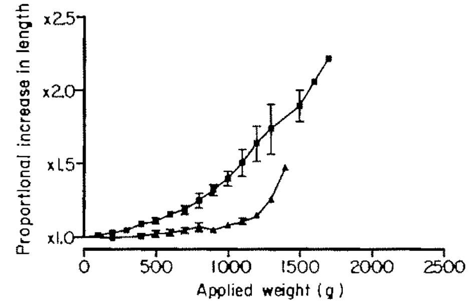

Fig. 3. Stretch curves of the 18-gauge catheters. Clear (⊢■⊣) and radio-opaque (⊢▲⊣). Means (SD).

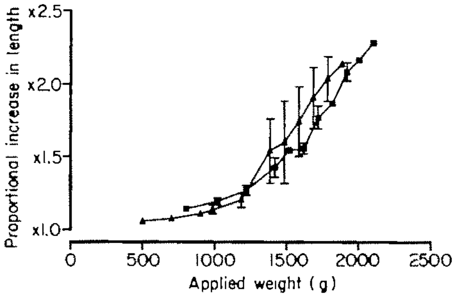

Fig. 4. Stretch curves of the 16-gauge catheters. Clear (⊢■⊣) and radio-opaque (⊢▲⊣). Means (SD).

The curves for the two 16-gauge catheters (Fig. 4) are similar at weights over 1200 g, although the radio-opaque curve lies significantly lower than the clear at 1000 and 1200 g ($p < 0.025$). The curves for the 18-gauge and 16-gauge radio-opaque catheters are not significantly different, but the 18-gauge clear catheter stretches more than its 16-gauge counterpart at all values above 800 g ($p < 0.05$).

Stretch before breaking

The proportional increase in length before breaking depends both on the elasticity of the catheter and its breaking strain. Results are summarised in Table 2. The distribution of stretch before breaking is noticeably skewed, and ranges rather than standard deviations are quoted to demonstrate this. The skew distribution arises because of the fact, previously noted, that breakages occur mainly on the rapidly curving, inelastic portions of the stretch graphs.

The 18-gauge radio-opaque catheter, because of its low breaking strain and limited stretch response, has a significantly lower stretch before breaking ($p < 0.005$) than any of the other catheters tested. The 16-gauge radio-opaque catheters stretched more before breaking ($p < 0.05$) than any other group. No other differences between groups achieved significance at the 0.05 level.

Table 2. Stretch before breaking.

Catheter/needle	Range	Mean	Median	
18-gauge radio-opaque	1.02–1.48	1.13	1.08	$p < 0.005$
18-gauge clear	1.30–2.22	1.63	1.51	
16-gauge clear	1.54–2.28	1.79	1.59	$p < 0.05$
16-gauge radio-opaque	1.66–2.52	2.11	2.14	

Figures given are lengths immediately before breaking expressed as multiples of the original lengths. Significance values on the right relate to the difference between the groups bracketed (Wilcoxon test).

Discussion

Efforts to reproduce the presumed clinical circumstances of catheter division failed because of the difficulty encountered in preventing catheters sliding back through the needle undamaged. This, in itself, is some indication of the robustness of the catheters tested. That the needle bevel has a role in dividing the catheters in the test situation is shown by the fact that all breakages occurred at the point where the catheter crossed the bevel.

The 18-gauge radio-opaque catheter and needle combination distinguishes itself from the other Minipacks tested by a low breaking strain and small amount of stretch before breaking. A tendency for a catheter to stretch without breaking may be considered an advantage, since it may protect the catheter against breakage if the applied force is transient. A further disadvantage of the radio-opaque catheter is that its opacity may prevent easy recognition of returning blood or cerebrospinal fluid.

It seems contradictory, therefore, to use a catheter which is more easily severed for the sole reason that the broken fragment can be easily located afterwards. Indeed, some authors[8,9] have noted that, despite their radio-opacity, fragments of catheter may still be impossible to detect radiographically against a background of vertebral bone, and that location on an X ray is no guarantee of location at laminectomy. Bromage[8] therefore suggests that the best course of action if a section of catheter is left in the patient, is to leave it alone unless symptoms develop. Several such cases have been followed up for periods of months or years without ill effect.[9-13] It is therefore suggested that the disadvantages of the Portex 18-gauge Minipack with radio-opaque catheter outweigh its sole advantage.

The 16-gauge radio-opaque catheter, however, compares well with its clear equivalent in all the factors tested here. With the exception, then, of the 18-gauge radio-opaque catheter, breaking strains of between one and two kilograms and a tendency for catheters to stretch by more than 30% of their lengths before breaking should be reassuring to all anaesthetists who have, for whatever reason, pulled the catheter back through the needle.

Acknowledgments

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Anaesthesia, 1987, Volume 42, pages 186–189

Forum

Explosive anaesthetic agents. A regional survey on the use of ether and cyclopropane

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Summary

A total of 156 consultant anaesthetists in the North East Thames Regional Health Authority were circulated with a questionnaire on their personal use of ether and cyclopropane and their attitudes towards restriction or withdrawal of these agents; 144 completed questionnaires were received (92% response). Four anaesthetists (3%) claimed to use ether regularly at least once a month and 27 anaesthetists (19%) felt that their anaesthetic practice would suffer if ether were withdrawn. Cyclopropane had 62 regular users (43%) and 65 anaesthetists (45%) felt that their practice would suffer if it were withdrawn. Ninety-two anaesthetists (64%) were prepared to confine their use of explosive agents to either the anaesthetic room or the operating room; of these, three-quarters favoured the anaesthetic room. There were few notable differences in the pattern of use of explosive agents between anaesthetists with more than 21 years in the specialty and those with less than 21 years.

Key words

Explosions.

In the United Kingdom, ether and cyclopropane are the only currently used anaesthetic agents which have a significant explosive potential. The risks of an explosion are small, but they are real and when an explosion does occur it is likely to kill the patient and injure operating theatre staff. A recent hazard notice from the DHSS[1] describes an incident where a 78-year-old patient undergoing minor nose surgery died after the anaesthetic gases which contained ether were ignited by the cautery. The force of the resultant explosion hurled operating room staff across the floor.

We wanted to study the use of the explosive anaesthetic agents in the North East Thames Regional Health Authority in order to answer four questions: how widespread is the use of ether and cyclopropane; would anaesthetic practice suffer if these agents were withdrawn; would anaesthetists be prepared to confine their use of explosive anaesthetic agents to the anaesthetic room or the operating theatre; is the pattern of use of the explosive agents different among the newer consultant anaesthetists (that is, those with less than 21 years' experience of anaesthesia) as compared with the more established consultants (those with more than 21 years in the specialty)?

Method

A questionnaire was circulated to 156 consultant anaesthetists in the North East Thames Regional Health Authority in February and March 1986. Replies received after 4 April 1986 were not included in our results. The questionnaire was kept brief and simple to encourage compliance and was estimated to have taken 1–2 minutes to complete. The format of the questionnaire is shown in Table 1.

The completed questionnaires were divided into two groups. One group comprised replies from consultants with 20 years or less spent in anaesthesia and the other group comprised replies from consultants with over 21 years' experience in anaesthesia. This divided consultants into two numerically equal groups, the newer consultants (NC) and the more established consultants (MEC). Forms which did not reveal the consultant's experience were put to one side and their results

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Table 1. Questionnaire used in present study.

Section A

(1) What is your grade?
(2) How many years have you spent in anaesthesia?

Section B

(1) Have you ever used ether? YES/NO
(2) Have you used ether in the last year? YES/NO
(3) Do you use ether at least once a month on average? YES/NO
(4) Would your anaesthetic practice suffer if you were unable to use ether at all? YES/NO
(5) Do you feel strongly that you should be able to use ether in every single anaesthetic room and operating theatre in which you are likely to give an anaesthetic? YES/NO

Section C

The same questions were asked as in section B but cyclopropane was substituted for ether.

Section D

(1) Would you be prepared to confine your use of both ether and cyclopropane to either the anaesthetic room or the operating theatre? YES/NO
(2) If so, which? ANAESTHETIC ROOM/OPERATING THEATRE

Section E

If you regularly use cyclopropane or ether, please list overleaf the occasions or surgical procedures for which you consider them to be the agents of choice.

included in the overall figures which appear in each table.

The findings were tabulated for the NC and MEC and analysed statistically (Chi squared test) to see whether there were any significant differences between the two groups. Comments were invited from regular users about the indications for using cyclopropane and ether. The comments received were all noted and recorded under general headings where appropriate.

Results

Experience of respondents. The range of experience was from 7.5 to 43 years. Seventy-one respondents had less than 21 years' experience in anaesthesia and 71 had over 21 years' experience. Two of the 144 respondents declined to enumerate their years in anaesthetic practice.

Use of ether. Of the 144 consultants who replied, 143 had used ether (see Table 2). Consultants who had not used ether during the last year were considered to be non-users. Sixty-two percent (44) of MEC and 91% (64) of NC were non-users. Overall, 76% were non-users. Consultants who did not use ether once a month on average, but who had used it within the last year, were considered to be occasional users.

Use of cyclopropane. All the anaesthetists questioned had used cyclopropane (see Table 3). The definitions of 'non-user', 'occasional user' and 'regular user' are as for ether.

Attitudes towards restriction of use. These are summarised in Table 4.

Indications for use of ether and cyclopropane. Indications for the use of these agents are shown in Tables 5 and 6. The main role for diethyl ether, apart from the relief of severe bronchospasm, seems to be in paediatric anaesthesia. Similarly, cyclopropane is considered a suitable induction agent for babies and children by 43% (62). The other main indication for cyclopropane appeared to be the high-risk patient, particularly in partial exsanguination.

Discussion

Anaesthetic explosions are very rare and fatalities from anaesthetic explosions are even rarer.[1,2] However, every explosion causes great concern, out of all

Table 2. Ether: usage and attitudes towards withdrawal and availability.

	More established consultants ($n = 71$)	Newer consultants ($n = 71$)	Overall ($n = 144$)
Non-users	44 (62%)	64 (91%)	110 (76%)
Occasional users	24* (34%)	6* (8%)	30 (21%)
Regular users	3 (4%)	1 (1%)	4 (3%)
Practice would suffer if withdrawn	14 (20%)	13 (18%)	27 (19%)
Should be available in every operating room and anaesthetic room	12 (17%)	11 (15%)	23 (16%)

* Significant difference ($p < 0.01$).

Table 3. Cyclopropane: usage and attitudes towards withdrawal and availability.

	More established consultants ($n = 71$)	Newer consultants ($n = 71$)	Overall ($n = 144$)
Non-users	35 (49%)	36 (51%)	72 (50%)
Occasional users	1* (2%)	8* (11%)	10 (7%)
Regular users	35 (49%)	27 (38%)	62 (43%)
Practice would suffer if withdrawn	32 (45%)	13 (18%)	65 (45%)
Should be available in every operating room and anaesthetic room	19 (27%)	25 (35%)	44 (31%)

* Significant difference ($p < 0.02$).

Table 4. Explosive agents: attitude towards restriction.*

	More established consultants	Newer consultants	Overall
Would confine use to anaesthetic room or operating room	38 (54%)	52 (73%)	92 (64%)
Would NOT confine use to anaesthetic room or operating room	25 (35%)	17 (24%)	42 (29%)
Did not reply	8 (11%)	2 (3%)	10 (7%)
Would restrict to anaesthetic room	25	43	69 (48%)
Would restrict to operating room	13	9	23 (16%)

* There was no significant difference between the newer and more established consultants.

Table 5. Indications for the use of ether in anaesthesia.*

Relief of severe bronchospasm (21)
Teaching (8)
Paediatric ENT surgery (7)
General paediatric anaesthesia (6)
ENT surgery: adults and children (4)
Induction of neonates (3)
Maintenance of anaesthesia following failed intubation (3)
Caesarean section following failed intubation (3)
Paediatric circumcision (2)
Paediatric endoscopy for foreign body (2)

Also: blind nasal intubation (1); as an alternative to muscle relaxants (1); cardiac tamponade (1); all general anaesthetics (1); as an alternative volatile agent in the presence of cardiac dysrhythmia (1).

* Figures in parentheses following each indication represent the numbers of respondents listing that indication.

Table 6. Indications for the use of cyclopropane in anaesthesia.*

Induction of children and babies (62)
Induction (and maintenance sometimes) of high-risk patients (18)
Induction of adults with poor veins (2)
Induction in the presence of obstruction of the upper airways (2)
Induction of urgent Caesarean section (2)
Minor operations in unpremedicated patients (1)
Myotonia and myasthenia (1)

* Figures in parentheses following each indication represent the numbers of respondents listing that indication.

proportion to its epidemiological significance both locally and nationally, and encourages a review of the practice of using potentially explosive anaesthetic agents.

It is possible that the use of explosive anaesthetic agents will be curtailed following the latest explosion.[2] It is even conceivable that the explosive agents will be withdrawn from use entirely. From the results of this survey, such a withdrawal would seriously inconvenience the 50% of anaesthetists who use explosive agents either regularly or occasionally. In the longer term it is likely that those anaesthetists will adopt the techniques of their colleagues who do not use explosive agents. It is most improbable that any current anaesthetic agent is completely indispensable.

Apart from the virtual elimination of the risk of anaesthetic explosions, there are potential financial savings. In the recently built theatre suite at Colchester General Hospital, the antistatic flooring cost £90 per square metre as opposed to a cost of £9 per square metre for non-antistatic flooring of similar appearance. Ordinary flooring would have allowed a saving of over £20 000 on the construction of four new operating rooms. In addition to savings on construction costs, we estimate that the ending of antistatic precautions would result in the saving of several hundred pounds a year in the regular checking of the efficacy of antistatic precautions.

However, complete withdrawal of the explosive agents would involve the loss of two agents which have unique characteristics. Teaching practice would suffer. Ether is cheap and safe and has a deservedly popular place in developing countries, where it can be easily administered through simple equipment such as the EMO vaporizer. It would be regrettable if British trained anaesthetists travelling to Third World countries were ignorant of ether. Cyclopropane has physical and pharmacological properties which make it a rapid and safe induction agent for babies, children and high-risk patients. Other techniques exist for the induction of such patients, but there is no other agent with similar properties. Its loss would therefore leave a gap in the anaesthetist's armamentarium.

Limitation of use would be more acceptable to the anaesthetic profession than complete abolition, although 29% of anaesthetists were not prepared to confine their use to either the anaesthetic room or operating room. Seventy-five percent of those accepting restricted use would prefer the anaesthetic room. No doubt this reflects the popularity of cyclopropane as an induction agent only.

It is likely that the present survey reflects rational practice, although the practice of using cyclopropane for induction of anaesthesia in children at the Hospital for Sick Children, Great Ormond Sreet, London, may be followed in the Thames Region more than in other Regions. If limitations were imposed on the use of the explosive agents, the following proposal might be acceptable to the majority of regular users: one anaesthetic room and one operating room in each suite to be set aside for use of explosive agents; cyclopropane to be available for induction in every anaesthetic room, but not to be used in operating rooms other than the one designated.

It is debatable whether these restrictions would do much to limit the risk of explosions, but they might act as a palliative to those opposed to the widespread use of explosive agents. It is probable that the current use of the explosive agents will change very little, since there is unlikely to be universal agreement on the form of any limitations.

Acknowledgments

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Isoflurane for anaesthesia in the dental chair. A comparison of the incidence of cardiac dysrhythmias during anaesthesia with halothane and isoflurane

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Summary

A randomised single-blind trial was performed to compare the effects on cardiac rhythm of isoflurane and halothane in 100 dental outpatients having extractions under general anaesthesia in the dental chair without tracheal intubation. The incidence of supraventricular dysrhythmias did not differ significantly. No patient in the isoflurane group developed a ventricular dysrhythmia, whilst nine of those who received halothane did so ($p = 0.0013$). The quality of anaesthesia was acceptable in the isoflurane group, but induction time was longer ($p < 0.05$) and the maximum heart rate was faster ($p < 0.01$). There was no difference in the maximum end tidal carbon dioxide concentrations measured in a further 20 cases.

Key words

Anaesthetics, volatile; halothane, isoflurane.
Complications; cardiac dysrhythmias.

In the United Kingdom more general anaesthetics are given for exodontia than for any other single operation. The majority are given on an outpatient basis in the dental chair with a mouth pack and nasal mask, but without a tracheal tube. Anaesthesia with halothane for dental extractions is known to be associated with a high incidence of cardiac dysrhythmias.[1-3] Isoflurane is known to be associated with a lower incidence of dysrhythmia in other branches of surgery[4] and in patients whose tracheas were intubated for outpatient oral surgery.[5,6] We compared its effects on cardiac rate and rhythm to that of halothane in patients receiving a general anaesthetic without tracheal intubation in the dental chair.

Methods

One hundred unpremedicated outpatients (ASA Grade 1 or 2) presenting to the Dental Hospital for exodontia under general anaesthesia were randomly allocated to receive either halothane or isoflurane. Any patient for whom either agent was contraindicated was excluded

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from the study. All the anaesthetics were administered in a standard manner by dental students, directly supervised by a consultant anaesthetist (R.S.E.), using a technique similar to that described previously.[7] The procedure was performed with the patient sitting upright. Induction of anaesthesia was normally inhalational for children and intravenous (methohexitone) for adults. Maintenance of anaesthesia was with 30% oxygen in nitrous oxide, supplemented with 1.5% isoflurane or 1.0% halothane (intended to give approximately equal inspired MAC levels), presented via a McKesson nasal inhaler with an expiratory valve modified for scavenging. Where clinically indicated, the concentration of halothane or isoflurane was increased to two or three times the initial one. Gases were provided by a Boyle's machine with a temperature-compensated vaporizer (Mark 3 Fluotec or Fortec, Cyprane Ltd) which had been calibrated by the manufacturer and checked by ourselves with an Engström Emma analyser. When anaesthesia was judged adequate, a mouth gag was inserted, the mouth packed and surgery allowed to proceed.

Standard lead I ECG electrodes were placed on the arms before induction. The cardiac rhythm and rate were monitored continuously on an ECG monitor with a digital readout of heart rate and recorded by the second investigator who was unaware of the inhalational agent being used. Any rhythm disturbance (defined as more than two non-sinus beats in any minute) was recorded; the rhythm was not disclosed to the administrator. A hard copy was made of any rhythm disturbance for analysis following surgery. The times of induction, the first extraction and recovery from anaesthesia (defined as when the patients first opened their eyes to command) were also recorded.

In order to establish whether the anaesthetic agents were associated with different end tidal carbon dioxide levels, a further 20 patients were randomly allocated to receive either halothane or isoflurane in exactly the same way as in the main study. The tidal carbon dioxide concentration was measured with an infrared gas analyser (Datex Normocap) using a catheter passed into the nasopharynx through the nose. The maximum end tidal carbon dioxide concentrations were recorded. The results were analysed with an unpaired *t*-test, Chi squared or Fisher's exact test, as appropriate.

Results

The two groups were indistinguishable with regard to age, sex distribution, number of extractions, number receiving intravenous induction and number of patients requiring greater than 'baseline' inspired vapour concentration (see Table 1). This table also shows that the mean time from induction to the start of the first extraction was one minute longer in the isoflurane group ($p < 0.05$). The total anaesthetic time from induction was also prolonged by 0.8 minutes in the isoflurane group but this did not achieve statistical significance.

Table 1. Age, sex, number of extractions, intravenous inductions, patients receiving greater than baseline inspired vapour concentration, mean time in minutes (SD) from induction to commencement of extraction, and total anaesthetic time for the two groups.

	Halothane ($n = 50$)	Isoflurane ($n = 50$)
Age (years): mean (range)	15 (2–56)	16 (2–57)
Sex: male/female	33/17	25/25
Extractions: mean (range)	4.0 (1–23)	3.4 (1–8)
Intravenous induction	18	19
Above baseline inspired vapour concentration	19	19
Induction–extraction, minutes	4.9 (1.5)*	5.9 (2.3)*
Induction–awakening, minutes	11.7 (5.0)	12.5 (4.7)

* Significant difference ($p < 0.05$).

Table 2. The incidence of dysrhythmias and maximum and minimum mean heart rates (SD) in the two groups.

	Halothane	Isoflurane
Dysrhythmia		
Supraventricular	9	6
Ventricular	9*	0*
Ventricular and supraventricular	2	0
Total with dysrhythmia	16†	6†
Mean heart rate, beats/minute		
Maximum	130 (17.9)‡	140 (14.5)‡
Minimum	82 (16.9)	84 (14.5)

* Significant difference ($p = 0.0013$).
† $0.05 > p > 0.01$.
‡ $0.01 > p > 0.001$.

Table 2 shows the maximum and minimum heart rates and incidence of dysrhythmias in the two groups. The incidences of supraventricular dysrhythmias (nine patients in the halothane group and six who received isoflurane) were not significantly different. No patient in the isoflurane group had a ventricular dysrhythmia, but nine of those in the halothane group did. This difference is significant ($p = 0.0013$). The mean age of the patients who developed ventricular dysrhythmias was greater and they had fewer extractions with a shorter operation time than those who developed supraventricular dysrhythmias. Two-thirds required greater than 1% inspired halothane concentration. In contrast, only a quarter of those who developed supraventricular dysrhythmias required a greater than baseline inspired vapour concentration.

All ventricular dysrhythmias were associated with surgical stimulation (in eight patients they occurred within 1.5 minutes after starting the first extraction and in the ninth after insertion of the gag). In two of the patients with supraventricular dysrhythmias, no stimulation had occurred; the timing of these was also more variable. One child (in the halothane group) developed 2:1 AV block in the recovery period, just before opening his eyes.

The mean maximum heart rate of 140 beats/minute for isoflurane was significantly greater than that of 130 for halothane ($p < 0.01$), but the minimum rates of 84 and 82 respectively were similar. No significant difference was found between the end tidal carbon dioxide levels in the two groups (see Table 3).

Table 3. End tidal carbon dioxide (kPa) for the 20 patients in whom this was studied.

	End tidal CO_2	
	Halothane ($n = 10$)	Isoflurane ($n = 10$)
Peak CO_2: mean (range)	5.1 (4.5–6.5)	5.3 (4.4–6.0)
CO_2 at extraction: mean (range)	4.7 (4.0–6.5)	4.9 (4.0–6.0)

Discussion

The 32% incidence of dysrhythmias with halothane during dental extraction in this study is similar to that from other centres[1,2,5] and compares with 12% in the isoflurane group. This difference was accounted for by an absence of ventricular dysrhythmias with isoflurane; the reduction in supraventricular dysrhythmias (12% with isoflurane versus 18% with halothane) does not reach significance. Dysrhythmias during exodontia are associated with hypoxia, hypercarbia, a light level of anaesthesia during extraction, an inhalational induction and the use of atropine. In common with others,[5] in this study we have not shown a greatly increased end tidal carbon dioxide concentration in patients receiving halothane and previous work from this department demonstrates that a 30% inspired oxygen concentration is not associated with severe hypoxic episodes.[7] Two-thirds of our patients who developed ventricular dysrhythmias were among the nineteen who required a greater than 1% inspired halothane concentration, but their operations were neither longer nor more difficult than those who remained in sinus rhythm. In contrast, none of the 19 patients who required greater than baseline inspired isoflurane developed a ventricular dysrhythmia. Although clinically there were no problems with isoflurane as an anaesthetic, the average maximum heart rate was higher and it took one minute longer to attain an adequate level of anaesthesia; this accords with experience elsewhere.[8–11]

Each year in the United Kingdom about three-quarters of a million anaesthetics are given to patients in the dental chair, frequently with halothane as the inhalational agent, usually without ECG monitoring. There is a low but consistent mortality in this group of fit, usually young patients. The cause of these deaths is not clear,[1,12] but at least some of them are due to sudden cardiovascular collapse during the anaesthetic and recovery period. Some of these collapses are thought to be due to vagal activity and the unrecognised faint and may not be affected by using isoflurane rather than halothane. However, cardiovascular collapse due to ventricular dysrhythmia should be markedly reduced by the use of isoflurane. It might be argued that the higher maximum heart rate with isoflurane is potentially harmful in patients with severe heart disease, but these patients would not normally be selected for outpatient dental anaesthesia. Although enflurane is also associated with a lower incidence of dysrhythmias in oral surgery[5,13] and in cases without tracheal intubation,[14] it is contraindicated in patients with a history of epilepsy[15] and has no demonstrable advantages (except cost) over isoflurane. Isoflurane may have a place in improving the safety of routine outpatient dental anaesthesia.

Acknowledgments

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Bradycardia associated with the use of vecuronium. A comparative study with pancuronium with and without glycopyrronium

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Summary

One hundred and twenty patients undergoing anaesthesia for elective surgery received either pancuronium or vecuronium for muscle relaxation. Within each of these two groups, half were given glycopyrronium and the remainder an inert placebo. The incidence of bradycardia or bradydysrhythmias was higher in the group having vecuronium compared with those given pancuronium. Glycopyrronium afforded protection against undesirable vagal activity.

Key words

Complications; bradycardia.
Neuromuscular relaxants; pancuronium, vecuronium.

Recently there have been a number of reports in the literature of bradycardia and bradydysrhythmias occurring during anaesthesia which involved the use of the newer non-depolarising neuromuscular blocking drugs, atracurium and vecuronium.[1-4] In all instances, a pre-anaesthetic anticholinergic drug had not been given. To date, there are no published studies in which the incidences of bradycardia and bradydysrhythmias associated with the use of vecuronium have been compared with those that appear when muscle relaxants such as alcuronium or pancuronium (which possess intrinsic vagolytic activity[5,6]) are used. Therefore, a study was designed to evaluate this and to determine whether anticholinergic administration could modify the incidence of bradycardia and bradydysrhythmias.

Methods

One hundred and twenty patients, ASA grades 1 and 2, of either sex, aged 15–70 years and with maximum weights of 100 kg (males) and 80 kg (females), undergoing general elective surgery, were included in the investigation. Patients with known cardiovascular disease were excluded, as were those who on the pre-anaesthetic 12-channel ECG showed evidence of cardiographic abnormalities. Similarly, patients taking drugs known to influence the cardiovascular system were not admitted into the study.

The patients were allocated, at random, into the following four groups of 30 each: group 1, pancuronium–saline; group 2, pancuronium–glycopyrronium; group 3, vecuronium–saline; group 4, vecuronium–glycopyrronium. At noon on the day before operation, the patients were visited by one of the investigators and oral consent to participate in the study was obtained. Those individuals who showed undue anxiety were given, during that afternoon, chlordiazepoxide 5–20 mg in two oral doses. In the evening, all patients received nitrazepam 5–10 mg.

Forty-five to 60 minutes before the induction of anaesthesia, the patients were given diazepam 5–15 mg orally together with oxycodone 0.12–0.14 mg/kg intramuscularly. On arrival in the operating theatre, an intravenous infusion of Ringer's solution was started and neuromuscular blockade monitored using the train-of-four pattern of stimulation with surface electrodes applied to the same arm. All drugs were injected into the intravenous infusion. Arterial blood pressure and heart rate were recorded using an automatic oscillotonometer from the contralateral arm. Following pre-oxygenation, the patients received either glycopyrronium or sodium chloride 0.9% injection of an equal volume in a double-blind manner. The dose of glycopyrronium was related to body weight as follows: $\leqslant 59$ kg, 0.20 mg; 60–74 kg, 0.25 mg; $\geqslant 75$ kg, 0.30 mg. Thiopentone sodium (2.5%) was given in a dose of 4.5–5.0 mg/kg over 30 seconds. On loss of the eyelash reflex, either pancuronium or vecuronium (in equal volumes) was injected in a dose of 0.1 mg/kg under double-blind conditions and the neuro-

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stimulator switched on. The patient's lungs were ventilated by a mask with nitrous oxide and oxygen (1:1) to maintain an end tidal carbon dioxide concentration of between 4.5 and 6%. When the second of the train-of-four contractions became undetectable,[7] the trachea was intubated. The patient's lungs were then mechanically ventilated with nitrous oxide and oxygen (2:1) supplemented with isoflurane 0.2–1.0%. Fentanyl was given in increments of 0.025–0.1 mg, as needed. Muscle relaxation was maintained with increments of 0.025 mg/kg of the appropriate drug, still under double-blind conditions.

The ECG was monitored continuously and recordings were made before and after the injection of any drug, at any time significant changes in heart rate occurred and when dysrhythmias appeared. Heart rate and blood pressure were recorded at 5-minute intervals except during any of the above events, when the device was cycled at one-minute intervals. A syringe containing atropine 1 mg was always on hand, and 0.5 mg was administered in all cases when the heart rate fell to 45 beats/minute or when, in the opinion of the anaesthetist, the clinical condition of the patient warranted such intervention.

Residual neuromuscular blockade was antagonised, when two of the visual train-of-four contractions had returned, with 1.5–2.5 mg neostigmine and 0.4–0.6 mg glycopyrronium. In the recovery room, the patients' heart rates and blood pressures were monitored and recorded for a minimum of 90 minutes. Atropine was given in situations similar to those stated earlier.

Heart rate and blood pressure data within the study groups were analysed with the paired *t*-test. The data between the groups were analysed using the Mann–Whitney test, while the nonparametric data were analysed by the Chi squared test. Values of p less than 0.05 indicated statistical significance. The study had the approval of the institutional ethics committee.

Results

Table 1 gives details of the patients in the four study groups. There were no statistically significant differences in any of the variables except for the total dose of muscle relaxant, which was higher in the two groups that received vecuronium ($p < 0.001$). There was no difference in the types of surgery between the four groups. The majority of operations were in the broad group consisting of abdominal, anal, genital and inguinal surgery. The remaining procedures involved breast, orthopaedic (excluding the use of tourniquet) and thyroid surgery.

Table 2 shows the heart rates before the start of anaesthesia, which were similar in all the four study groups, as were the lowest heart rates recorded in the recovery room. Heart rates recorded 10 minutes following tracheal intubation (that is, during maintenance of anaesthesia) revealed that those patients who had received vecuronium had lower heart rates than those given pancuronium ($p < 0.001$). Administration of glycopyrronium did not significantly affect heart rate, irrespective of the relaxant used. However, the minimum heart rates recorded during surgery were consistently lower in the two groups of patients who had been given vecuronium. Within each study group, the lowest heart rate achieved was significantly lower than the control value ($p < 0.01$) except for group 2. Associated with the decrease in heart rate was a concomitant and significant ($p < 0.05$) fall in systolic and diastolic blood pressure. The difference in this respect between the relaxants is statistically significant ($p < 0.001$). In the patients who received vecuronium, the decrease in heart rate was significantly reduced ($p < 0.025$) in the individuals who had been pretreated with glycopyrronium compared with those who had been given saline.

Table 1. Patient and anaesthesia data for the four groups: values expressed as means (SD). There were 30 patients in each group.

	Group			
	1	2	3	4
Age, years	43	42	41	44
	(12)	(12)	(11)	(12)
Sex, number of males	9	13	13	15
Weight, kg	64	69	69	72
	(11)	(9)	(11)	(9)
Height, cm	166	170	168	172
	(9)	(4)	(7)	(8)
Total relaxant, mg	7.4	8.4	13.3	13.0
	(1.6)	(2.3)	(4.4)	(4.6)
Total fentanyl, μg	135	156	177	148
	(92)	(92)	(150)	(84)
Duration of anaesthesia, minutes	133	126	141	124
	(75)	(62)	(71)	(68)

Table 2. Mean (SD) heart rates (beats/minute) of the four groups at various times of the study.

	Group			
	1	2	3	4
Before anaesthesia, control	71	71	71	67
	(16)	(14)	(14)	(13)
Ten minutes after intubation	81	86	66	72
	(12)	(13)	(15)	(13)
Lowest during surgery	64	67	51	56
	(9)	(11)	(7)	(8)
Lowest during recovery	61	61	61	58
	(9)	(9)	(10)	(13)

The number of patients with a heart rate of 45 beats/minute or lower, who received peroperative atropine or who presented with dysrhythmias, is shown in Table 3. None of the patients who received pancuronium demonstrated bradycardia to this degree, nor did they require atropine. On the other hand, 13 patients who received vecuronium manifested bradycardia and one additional patient was given atropine because systolic blood pressure had fallen to 80 mmHg, with a heart rate of 47 beats/minute. The difference between the groups is statistically significant

Table 3. Numbers of patients within the four groups with bradycardia (≤45 beats/minute), dysrhythmias and requiring atropine intra-operatively.

	Group			
	1	2	3	4
Bradycardia	0	0	11	2
Dysrhythmias	0	2	7	5
Intra-operative atropine	0	0	12	2

with respect to both administration of atropine and incidence of bradycardia ($p < 0.01$).

In the two groups of patients who had been given saline (groups 1 and 3), significantly more atropine was administered in the group which had received vecuronium ($p < 0.01$). Notably, in the two groups pretreated with glycopyrronium (groups 2 and 4) the difference was minimal and statistically insignificant.

The predominant type of dysrhythmia recorded was junctional rhythm. In those patients who had not received the anticholinergic, vecuronium administration was related to a significantly greater number of patients with dysrhythmias ($p < 0.01$). There was, however, no significant difference between the two relaxants in the patients who had received glycopyrronium. In three instances when atropine was injected to correct serious (sinus) bradycardia, nodal rhythm developed immediately.

Discussion

This study suggests that the use of vecuronium without glycopyrronium often results in bradycardia. The use of routine anticholinergic drugs before anaesthesia, at least in the United Kingdom, has been decreasing.[8,9] This practice is, arguably, justifiable when muscle relaxants such as alcuronium or pancuronium are used which, as stated earlier, have intrinsic vagolytic activity. This particular side effect may be expected to protect the patient against excessive vagal tone mediated through the surgical procedure and/or concomitant drug therapy. On the other hand, the increase in heart rate produced for example by pancuronium may be deleterious, especially in patients with pre-existing cardiovascular disease.[10] Thus, agents such as atracurium, pipecuronium or vecuronium, which have a cleaner pharmacological profile, may be of value in such at-risk patients.[11] However, this advantage of these newer relaxants must be weighed against the possible risk of serious intra-operative bradycardia and bradydysrhythmias.

The anticholinergic glycopyrronium protected the greater proportion of patients against these adverse effects. The dose of glycopyrronium used was within the recommended range, but it is conceivable that a larger dose would have provided abolition of intra-operative bradycardia. It might be argued that the use of an anticholinergic would obviate one of the desirable features of vecuronium, namely, the lack of undue tachycardia; in the present study, the use of glycopyrronium was not accompanied by a significant rise in heart rate.

The mechanism of increased vagal activity is multifactorial and surgical stimuli and the use of vagotonic drugs are only two components. In spite of this, in the present work there was no relation between the total dose of fentanyl and the incidence of bradycardia (the types of surgery were similar in all four groups)

An interesting observation was the development of nodal rhythm following the administration of atropine to correct sinus bradycardia. This has been explained by Averill and Lamb[12] as being due to an early vagal action of atropine at the AV node.

In conclusion, the use of vecuronium without glycopyrronium is more liable to result in bradycardia. If vecuronium and, presumably, the other relaxants which lack intrinsic vagolytic activity are used, then perhaps the routine use of anticholinergic premedication (for example, atropine or glycopyrronium) should be reconsidered.

Acknowledgments

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Evoked potentials following diazepam or fentanyl

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Summary

The effects of fentanyl or diazepam on somatosensory, visual and brainstem auditory evoked potentials were studied in 13 healthy patients scheduled for elective surgery. Following control recordings of evoked potentials, either diazepam 20 mg or fentanyl 200 μg was administered intravenously. Evoked potentials were then recorded twice in the subsequent hour. No significant changes occurred in the latency or amplitude of somatosensory, visual or brainstem auditory evoked potentials. Although dose-related changes in evoked potential latencies and amplitudes have been demonstrated with both the inhalational and intravenous anaesthetics, these changes did not occur with diazepam or fentanyl used alone. An anaesthetic technique based on these two drugs would be suitable when intra-operative evoked potential monitoring is required to assess ischaemia and preservation of evoked responses.

Key words

Analgesics, intravenous; fentanyl.
Brain; evoked potentials.

A single stimulus, such as a light flash, evokes a small electrical response which can be recorded over the occipital cortex. This resultant potential is very small and is lost in the background electroencephalogram (EEG), which is of much higher voltage. If the stimulus is applied repetitively and the EEG is averaged in the period after stimulation, a compound evoked potential is built up and the non stimulus-related portion of the EEG is reduced below the level of background noise. Evoked potentials therefore test specific neural pathways, e.g. eye to occipital cortex. Measurements may be made of latency, the time from stimulus to the appearance of the potential (measured in milliseconds), and amplitude, from the first negative peak to the greatest succeeding positive valley (measured in μV). Since evoked potentials test the integrity of specific neural pathways, peroperative monitoring has been used to test for ischaemia from excessive retraction hypoxia or interference with blood supply; this aspect of evoked potential monitoring has been reviewed.[1]

In contrast to the electroencephalogram, where each anaesthetic agent produces different EEG changes, intravenous and inhalational agents both prolong the latency and decrease the amplitude of cortical evoked potentials.[2–4] Cortical evoked potential monitoring has therefore been suggested as a possible neurophysiological indicator of anaesthetic depth.[2] This study was designed to determine the effects of a benzodiazepine (diazepam) and an opioid (fentanyl) on somatosensory (SEP), visual (VEP) and brainstem auditory (BAER) evoked responses.

Methods

Following local ethics committee approval, thirteen ASA I patients (5 male, 8 female; mean age 33.6 years, SD 9.4 years) scheduled for elective surgery gave informed consent to the study. Evoked potentials were recorded using a Medelec Sensor with an Apple II microcomputer used for data storage. Silver/silver chloride electrodes were used and electrode impedance was monitored and maintained between 1 and 2 kΩ.

Somatosensory evoked potential (*SEP*). The median nerve was stimulated with a 100 μs constant current pulse generator, set at at least 150% of motor threshold at 2 Hz for 256 repetitions to record SEP. The electrodes were placed over the contralateral hand area and over the fifth cervical vertebra and these were both referred to a midfrontal reference. Filters were set at 0.5–1500 Hz.

Visual evoked potential (*VEP*). The visual stimulus for VEP was a flash from an SLE photic stimulator at 2 Hz for 128 repetitions. The electrode derivation used

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was 5 cm above the inion in the midline referred to a midfrontal reference. Filters were set at 0.5–250 Hz.

Brainstem auditory evoked responses (*BAER*). The auditory stimulus used for BAER was a 10 Hz square-wave click of 100 μs duration, delivered at 80 dB via headphones for 1024 repetitions. The electrode derivation used was from the ipsilateral mastoid to the vertex. Filters were set at 10–3000 Hz.

In order to facilitate peak identification, recordings were made in duplicate. Following baseline measurements, diazepam 20 mg or fentanyl 200 μg was administered intravenously as a bolus. Measurements of SEP, VEP and BAER were made during the first 30-minute period (period A) and the succeeding 30-minute period (B) after intravenous injection. Verbal contact was maintained with the patients throughout.

Measurements. A typical evoked potential and the measurements made on it are shown in Fig. 1. The latency is stated in milliseconds and refers to a time interval between the stimulus and the first negative peak on the evoked potential waveform. The amplitude of the evoked potential is measured in μV. In this study, we have measured amplitude from the first negative peak to the greatest succeeding positive valley. The potential difference between these two points is taken as the amplitude.

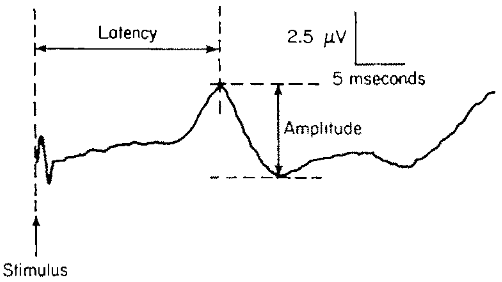

Fig. 1. Example of measurement techniques for a somatosensory evoked potential (SEP). The SEP shown represents the average of 200 repetitions. An upward deflection indicates a negative potential. The latency measurement is made from the time of stimulus to the first major negative peak and the amplitude measurement from that negative peak to the next positive valley.

The above measures of amplitude were used for SEP and VEP. In addition, for the SEP, central conduction time (CCT) was also measured. This is the time in milliseconds between the appearance of the first negative peak recorded over the fifth cervical vertebra and the negative peak recorded over the contralateral hand area of the cortex.

The brainstem auditory evoked response (BAER) is a more complex waveform than SEP or VEP. There are five generally recognisable waveforms which occurred within the first 10 milliseconds after stimulation and represented the passage of the signal from the ear to the brainstem. Wave I is thought to arise from the acoustic nerve, wave II from the cochlear nucleus, wave III from the superior olivary complex, wave IV from the lateral lemniscus and wave V from the inferior colliculus. In this study, we report the latencies of waves I, III and V of the BAER. We did not always obtain reliable data for waves II and IV and data from these waveforms are therefore not reported. Owing to variability within the trial, no amplitude measurements of BAER were made.

Results

Somatosensory evoked potential (*SEP*). Figure 2 shows SEPs after diazepam and fentanyl. During the hour after administration of the drugs, no alterations in SEP latency or amplitude were seen, except in subject 4 who showed a reduction in amplitude. Considering the group data (Table 1), there were no consistently measurable changes in SEP latency or amplitude, or central conduction time (CCT). Following diazepam, as shown in Fig. 2, there was an alteration in the later components of the SEP. In the control recording the later component was positive, i.e. a downward deflection, but after administration of diazepam this component was negative, that is, an upward deflection. This is probably related to the alteration in level of consciousness produced by diazepam.

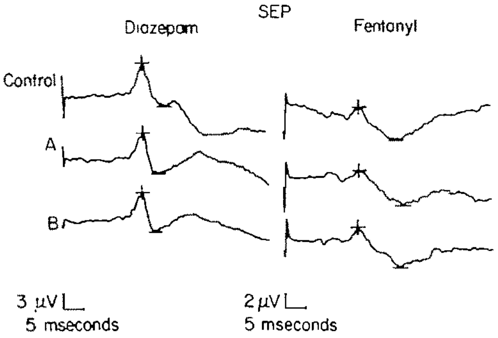

Fig. 2. Somatosensory evoked potential (SEP) recordings from one subject following diazepam and from one following fentanyl. Upward deflection indicates a negative potential. The stimulus was given at the left-hand side of the recording, and the initial large downward deflection indicates stimulus-related artifact. Averaging was continued for 100 milliseconds after the stimulus. Latencies were measured from the stimulus, at the left-hand side of the tracing, to the first vertical marker. Amplitude measurements were made between horizontal markers. 'A' indicates a measurement during the first 30 minutes after drug administration and 'B' the second 30 minutes.

Visual evoked potential (*VEP*). Figure 3 shows VEPs after diazepam and fentanyl. Following fentanyl, no change in latency or amplitude could be detected. Following diazepam there was no change in the latency of the first negative peak or the succeeding positive peak, although the amplitude decreased. The

Table 1. Somatosensory evoked potentials (SEP) and central conduction time (CCT) following diazepam 20 mg or fentanyl 200 μg. Data are mean (SD).

	Baseline	First 30 minutes after drug	Second 30 minutes after drug
Diazepam			
CCT, mseconds	6.1 (0.3)	5.9 (0.6)	5.9 (0.4)
Latency, mseconds	19.1 (0.8)	19.2 (1.0)	19.4 (1.1)
Amplitude, μV	6.3 (2.2)	5.2 (1.7)	4.5 (2.3)
Fentanyl			
CCT, mseconds	5.8 (0.3)	5.6 (0.3)	5.5 (0.3)
Latency, mseconds	19.0 (0.9)	19.0 (1.1)	18.9 (1.0)
Amplitude, μV	5.7 (2.2)	4.6 (1.4)	5.3 (2.3)

Table 2. Visual evoked potentials (VEP) following diazepam 20 mg or fentanyl 200 μg. Data are mean (SD).

	Baseline	First 30 minutes after drug	Second 30 minutes after drug
Diazepam			
Latency, mseconds	73.7 (14)	73.0 (6.0)	73.7 (2.5)
Amplitude, μV	15.6 (8.2)	16.9 (4.5)	17.7 (5.8)
Fentanyl			
Latency, mseconds	72.3 (3.5)	76.2 (4.8)	75.0 (5.4)
Amplitude, μV	19.0 (3.8)	19.2 (7.4)	20.0 (6.4)

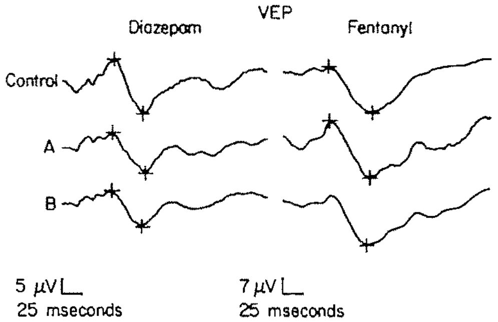

Fig. 3. Visual evoked potential (VEP) recordings from one subject following diazepam and from one following fentanyl. Upward deflection indicates a negative potential. The stimulus was given at the left-hand side of each tracing and averaging continued for 250 milliseconds. The latencies of the first major negative peak were measured from the stimulus, at the left-hand side of the tracing, to the first vertical marker, and amplitudes were measured between the horizontal markers. 'A' indicates a measurement during the first 30 minutes after drug administration and 'B' the second 30 minutes.

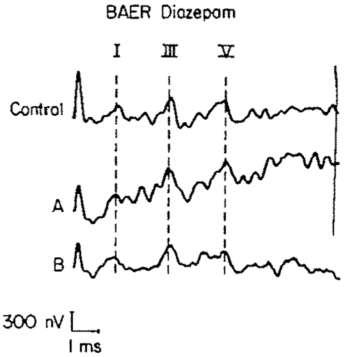

Fig. 4. Brainstem auditory evoked response (BAER) recordings from one subject following diazepam. An upward deflection indicates a negative potential. The stimulus was given at the left-hand side of the tracing at the marked upward-going stimulus artifact. Averaging was continued for 10 milliseconds after stimulus. The negative, upward, deflections of waves I, III and V are indicated by vertical lines. 'A' indicates a tracing obtained during the first 30 minutes after drug administration and 'B' the second 30 minutes.

group data (Table 2) show that this alteration in amplitude is not a consistent finding; the mean amplitude in patients given diazepam remained constant. Inconsistent changes occurred in individual patients.

Brainstem auditory evoked responses (BAER). Figure 4 shows BAERs after diazepam administration. No measurable changes in latency or amplitude occurred. Again, no consistent alterations in BAER latencies were found for the group as a whole (Table 3). During the first 30 minutes after fentanyl, BAER could only be measured in two patients and not at all during the second 30 minutes. These recordings were contaminated by muscle artefact as patients were unable to relax sufficiently for adequate potentials to be recorded.

Discussion

Evoked potential monitoring has two possible applications in anaesthesia. First, for monitoring peroperative ischaemic episodes in the brain and spinal cord,[1]

Table 3. Brainstem evoked potentials (BAER) following diazepam 20 mg or fentanyl 200 μg. No reliable data for the fentanyl subjects could be obtained in the second 30 minutes after drug administration. Data are mean (SD).

	Baseline	First 30 minutes after drug	Second 30 minutes after drug
Diazepam			
Wave I latency, mseconds	1.7 (0.1)	3.7 (0.1)	1.7 (0.1)
Wave III latency, mseconds	3.7 (0.1)	3.7 (0.1)	3.7 (0.1)
Wave V latency, mseconds	5.7 (0.1)	5.7 (0.1)	5.8 (0.1)
Fentanyl			
Wave I latency, mseconds	1.5 (0.1)	1.5 (0.2)	—
Wave III latency, mseconds	3.7 (0.2)	3.5 (0.2)	—
Wave V latency, mseconds	5.6 (0.1)	5.5 (0.2)	—

since marked prolongation of evoked potential latencies and reductions in amplitude occur with ischaemia. Second, evoked potentials may be useful as an index of anaesthetic depth.[2] For both uses it is important to establish the effects of drugs that are used before or during anaesthesia on evoked potentials.

Inhalational and intravenous anaesthetics both cause a dose-related increase in latency and a reduction in amplitude of the early cortical components of the auditory evoked response.[3,5] Halothane has been shown to produce an increase in the flash-evoked VEP.[6] Our own studies have shown that up to 50% nitrous oxide produces a dose-related reduction in VEP and SEP amplitude, with no change in latency.[4] Isoflurane produces dose-dependent increases in latency of SEP, VEP and BAER, with reductions in amplitude of SEP and VEP.[7] The cortical effects of anaesthetics are therefore similar, in that all agents studied tend to increase latency and reduce the amplitude of cortical potentials. Domino[8] studied the effect of sedatives and psychomotor stimulants on VER and found that morphine had no significant effect on VER and that scopolamine enhanced the response.

The findings of the present study are essentially negative, in that neither diazepam 20 mg nor fentanyl 200 μg produced any measurable effect on evoked potentials. Following diazepam, there was an alteration in the later component of the somatosensory evoked potential. In the awake state this tended to be positive, but following diazepam, this component altered in a variable manner, becoming negative in some cases. These later components of the SEP are generally variable and this alteration in morphology may reflect some degree of sedation and alteration of level of consciousness. The findings are important, however, because they show that relatively large doses of a benzodiazepine or opioid can be used without affecting evoked responses and should therefore not interfere with peroperative monitoring. From this study, we cannot exclude possible interactions with other anaesthetic agents and this area is worthy of further study. Brainstem auditory evoked responses are relatively resistant to the effects of anaesthetics. The volatile agents will produce very small increases in latencies,[3,7] but the BAER is not affected by etomidate[5] or fentanyl 70 μg/kg.[9] In this study, we were unable to record BAERs satisfactorily after fentanyl because the subjects were unable to relax sufficiently. This may be a manifestation of the muscle rigidity seen after larger doses of fentanyl.[10]

In conclusion, we have demonstrated that intravenous fentanyl 200 μg and diazepam 20 mg have no effect on evoked potentials. It may therefore be appropriate to use opioids or benzodiazepines for premedication when peroperative evoked potential monitoring is required.

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Vecuronium—A variable dose technique

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Summary

Vecuronium was administered to patients in doses of 100, 150, 200 and 250 µg/kg and the duration of action recorded by noting the time to recovery of the first twitch in a train-of-four to 10% of the control twitch. The recovery times ranged from a mean of 28.4 minutes (100 µg/kg) to 72.4 minutes (250 µg/kg). It is suggested that for operations of medium to long duration the use of a large initial bolus dose of vecuronium is a technique with advantages over the use of an infusion or frequent incremental doses.

Key words

Neuromuscular relaxants; vecuronium.

The advantages of the new neuromuscular blocking agent vecuronium (Norcuron), are the complete absence of any direct effect on the cardiovascular system, a relatively short duration of action, minimal cumulative effects following repeated doses and ease of reversal.[1–3] It does not cause the release of histamine.[4] However, in clinical practice, vecuronium is more difficult to use for operations of medium or long duration than longer-acting drugs. The rapid recovery necessitates constant monitoring to anticipate recovery of neuromuscular activity so that supplementary doses may be administered before muscle tone returns. In order to achieve good relaxation for long operations, frequent top-up doses or a continuous infusion of drug is required.

The use of vecuronium as an infusion, following a bolus dose, as described by Noeldge *et al.*[5] and Newton,[6] requires an infusion pump and a separate intravenous line as well as a neuromuscular block monitor. Less than 100% block must be maintained and should either the intravenous line, the infusion system or the monitor fail for any reason, the technique has to be abandoned. It has been suggested that because infusion techniques increase the complexity of the anaesthetic technique, they may also reduce safety.[7]

As vecuronium is highly specific in its action at the neuromuscular junction,[8] it is free from side effects, even when administered in very large doses. It is reasonable to utilise this large safety margin of vecuronium, which results from the differential between the dose required to produce neuromuscular block and that producing any cardiovascular effect,[9–11] by administering larger than paralysing doses of the drug in order to prolong the duration of its activity. This would simplify the use of this drug for surgery of medium and longer duration. To this end a study was made of the duration and recovery times following neuromuscular block induced by the administration of 100, 150, 200 and 250 µg/kg vecuronium to patients undergoing surgery under controlled conditions.

Method

Fifty-seven patients, between 18 and 87 years of age, who presented for surgery necessitating muscle relaxation, were included in this study. Only patients with evidence of renal or liver disease were excluded. All patients were premedicated with either papaveretum 10–20 mg, or pethidine 50–100 mg and a vagolytic agent in an appropriate dose.

Induction of anaesthesia was carried out with thiopentone 4–6 mg/kg followed by vecuronium 100, 150, 200 or 250 µg/kg. All patients had their tracheas intubated and their lungs ventilated with nitrous oxide, oxygen and enflurane 0.6–1.0%. The ventilation was adjusted to give an end tidal carbon dioxide of between 4 and 5%.

The duration from the injection of the bolus dose of vecuronium to 10% recovery of the first twitch of a train-of-four, using electromyographic monitoring with the Datex Relaxograph, was recorded. This was taken to be the duration of surgical paralysis. When indicated (two twitches in a train-of-four), top-up doses of 30 µg/kg were given to patients who had received 100 or 150 µg/kg bolus doses and 50 µg/kg to those who

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had received 200 or 250 µg/kg doses. All patients received atropine 1.2 mg and neostigmine 2.5 mg at the end of the operation if spontaneous recovery to a T_4 ratio (ratio of the 4th to the 1st twitch) of less than 0.7 had occurred.

Where possible the recovery slope to 75% T_1 recovery (or as close as clinically realistic), was recorded and the recovery index (RI) calculated as the time from 25–75% recovery of first twitch of the train-of-four response measured by EMG activity. The investigation was carried out by two different anaesthetists and the results from two different centres were pooled to produce the results presented.

Results

Confidence in the results was enhanced by the close agreement between the results from the two different centres. The observations of the duration of action of vecuronium summarised in Table 1 and Figs 1 and 2, indicate that increasing doses of the drug predictably prolong the duration of the neuromuscular block produced; vecuronium therefore changes from a relatively short-acting drug to an intermediate or long-acting neuromuscular blocking agent. For a 100 µg/kg bolus injection, the mean duration to 10% recovery was 28.4 minutes (SD 4.2). This correlates well with other studies by Swen[12] and Baird *et al.*[13] The mean recovery index for the nine patients studied was 13.7 minutes (SD 3.7).

Table 1. Duration of action of vecuronium.

Dose (µg/kg)	Mean duration till 10% recovery (SD), minutes	Recovery index (SD), minutes
100	28.4 (4.2) (n = 10)	13.7 (3.7) (n = 9)
150	40.9 (6.2) (n = 19)	16.1 (6.5) (n = 17)
200	54.2 (11.2) (n = 15)	23.3 (7.4) (n = 15)
250	72.4 (12.2) (n = 13)	30.0 (11.8) (n = 13)

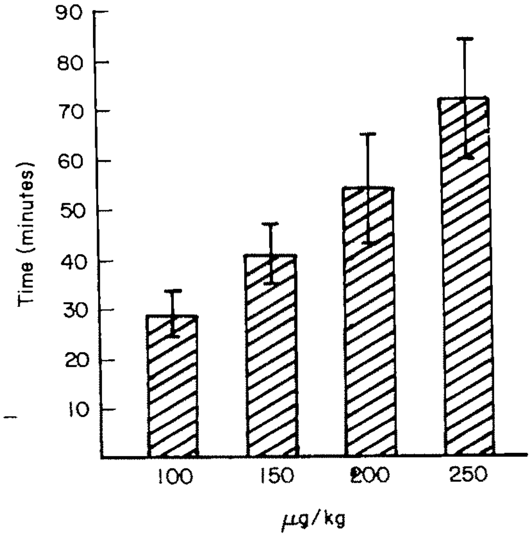

Fig. 1. Mean duration of surgical relaxation following 100–250 µg/kg vecuronium.

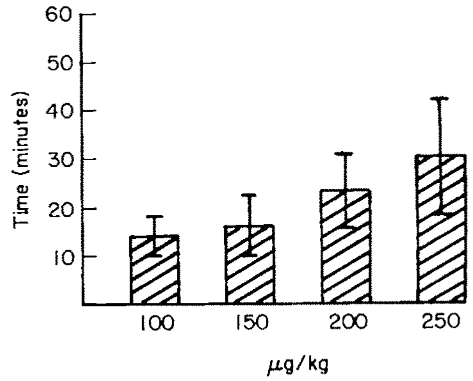

Fig. 2. Recovery indices (25–75% recovery of first twitch of train-of-four) following 100–250 µg/kg vecuronium.

Following the administration of 150 µg/kg, the mean duration of action in 19 patients was 40.9 minutes (SD 6.2) with recovery index of 16.1 minutes (SD 6.5) in 17 patients in whom evaluation was possible. When 200 µg/kg of vecuronium was administered, the mean duration of the block in 15 patients was 54.2 minutes (SD 11.2) with a mean recovery index of 15 patients evaluated as 23.3 minutes (SD 7.4). The 250 µg/kg dose of vecuronium gave a longer mean duration of action than the previous doses used in 13 patients; this was 72.4 minutes (SD 12.2) and the mean recovery index found in 13 patients was 30.0 minutes (SD 11.8).

All patients had full clinical neuromuscular recovery either before or after reversal with neostigmine and no signs of recurarisation occurred at any time. No significant changes in blood pressure or pulse rate attributable to the vecuronium occurred in any of the patients included in the study.

The results of the administration of the top-up doses were equally predictable. When a top-up dose of 30 µg/kg was administered following a bolus dose of either 100 or 150 µg/kg, the mean duration of effect was 21 minutes (SD 7.2). A 50 µg/kg top-up dose following a bolus dose of 200 or 250 µg/kg, gave a further period of paralysis (until just two twitches of the train-of-four response returned) of 33 minutes (SD 9.6) (Table 2).

Table 2. Duration of action of vecuronium with top-up doses.

Dose (µg/kg)	Top-up dose (µg/kg)	Duration of action (SD), minutes
100–150 µg/kg	(1) 30 µg/kg	21 (7.2) (n = 8)
	(2) 30 µg/kg	23 (7.8) (n = 6)
200–250 µg/kg	50 µg/kg	33 (9.6) (n = 6)

(1) 1st top-up dose; (2) 2nd top-up dose.

Discussion

Our investigation has shown that by adjusting the initial bolus dose of vecuronium it is possible to provide either a short, medium or long-acting degree of relaxation without cardiovascular side effects, thus fulfilling in one drug, the requirements suggested by Savarese and Kitz[14] for a range of short, medium and long-acting agents.

The duration of action of 100 μg/kg of vecuronium found in this study, 28.4 minutes (SD 4.2), compares closely with the reports of other investigators. Even in doses as high as 250 μg/kg vecuronium produces a response that is more predictable in its duration of action than either tubocurarine or pancuronium. Indeed, in a recent study, Swen[12] found that following 100 μg/kg pancuronium the duration of action was 74 minutes (SD 35). The very large variation in the duration of action and in the degree of block produced by pancuronium and tubocurarine is not seen with doses of vecuronium up to 250 μg/kg. Although there is a tendency for the duration of block to be less predictably prolonged in elderly or debilitated patients, this is not evident until doses in excess of 200 μg/kg are used. The recovery index (time for 25–75% recovery to occur) is more prolonged as the dose is increased. However, even at 200 μg/kg, it is only 23.3 minutes. This is considerably faster in our experience than after 100 μg/kg pancuronium or 400 μg/kg tubocurarine, which are required to give a similar duration of surgical relaxation under similar conditions.

In two elderly patients who received 250 μg/kg, the recovery index was unduly prolonged (41 and 46 minutes). It would appear that at this dosage there is near saturation of the immediate fast tissue distribution sites for the drug; this necessitates slower renal and hepatic elimination to lower the plasma concentration. This is in keeping with the findings of d'Hollander *et al.*[15] Although both these elderly patients recovered full neuromuscular transmission following neostigmine, it must be assumed that this represents the upper dosage limit for the safe administration of this drug, especially in elderly or debilitated patients.

These findings suggest that one can preselect a dose of vecuronium to produce either a short, medium or long-acting degree of neuromuscular blockade without cardiovascular side effects. In addition, the duration of action may be more predictable than with existing agents and with a more rapid spontaneous recovery than after either tubocurarine or pancuronium. The technique has significant advantages over other relaxant techniques, both in its simplicity and safety.

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Anaesthesia, 1987, Volume 42, pages 202–205

Postoperative headache in young patients after spinal anaesthesia

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Summary

Spinal anaesthesia was performed on 247 young adult patients with a 25-G needle. Rectal administration of indomethacin had no significant effect on the incidence of postdural puncture headache, which occurred in 16.8% of patients who received the drug compared to 24.5% who received a placebo. A history of headache pre-operatively did not influence the incidence of postlumbar puncture headache.

Key words

Anaesthetic techniques, regional; spinal.
Complications; headache.
Analgesics, nonnarcotic; indomethacin.

There has been a renewed interest in the use of spinal anaesthesia since the development of suitable new long-acting local anaesthetics and improved equipment.[1] The technique, however, suffers from the well known disadvantage of postdural puncture headache. This is especially a problem in young patients,[2] and we have recently found a 37.2% incidence in young male outpatients.[3] Some investigators report an incidence as low as 4% with the use of 25-G needles.[4]

Indomethacin given postoperatively has been reported to reduce the incidence of this headache. To investigate this further, a prospective study was undertaken in young hospitalised patients of either sex receiving spinal anaesthesia.

Methods

The test protocol was approved by the local ethical committee. Informed consent was obtained from 250 adult patients (less than 55 years old) of ASA groups 1 and 2 who were to receive spinal anaesthesia. The latter was performed using a 25-G spinal needle; following this they were randomly allocated in a double-blind manner to receive either indomethacin 100 mg or a placebo *per rectum* 4 hours postoperatively.

All the patients had been seen pre-operatively and a history of headache sought. Complaints of headache occurring more than once a week were regarded as a positive history. They were not informed of the special characteristics of a spinal headache.

The patients were interviewed by one of the investigators on days 1, 2 and 3 after surgery. If they had been discharged from hospital before day 3, they were contacted by telephone or letter. The type, severity and duration of the headache were recorded. Severity was measured using a visual analogue scale from 1–10. The headache was considered as being typically postspinal if it fulfilled the criteria laid down by Driessen:[6] occurred after mobilisation, aggravated by the erect or sitting position, relieved by lying flat, mostly occipital or frontal, accompanied by dizziness, vomiting, rigidity of the neck and visual disturbances.

Postspinal headache was initially treated with bed rest and analgesics. If not relieved within 2–3 days, an epidural blood-patch was considered.

For statistical evaluation of the data, the Chi squared test and Student's *t*-test were used.

Results

Three patients were lost to follow-up and their results are not included in the study. The indomethacin and placebo groups were comparable with regard to age,

Table 1. Sex, age and type of surgery in two groups of patients after spinal anaesthesia through a 25-G needle.

	Indomethacin (n = 125)	Placebo (n = 122)
Mean age (years)	34.2	33.3
Men:women	88:37	74:48
Surgery		
Orthopaedic	88	84
Perineal	20	17
Stripping	10	11
Other	7	10

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Table 2. Incidence of PDPH in two groups of patients receiving spinal anaesthesia.

Age group	Sex	Indomethacin ($n = 125$)	Placebo ($n = 122$)
18–29 years	M	9/46 (19.5%)	9/31 (29.0%)
	F	8/12 (66.7%)	8/14 (57.1%)
30–39 years	M	1/20 (5.0%)	3/22 (13.6%)
	F	1/10 (10.0%)	4/16 (25.0%)
40–58 years	M	2/22 (9.1%)	2/21 (9.5%)
	F	0/15 (0.0%)	4/18 (22.2%)
All patients	M	12/88 (13.6%)*	14/74 (18.9%)*
	F	9/37 (24.3%)**	16/48 (33.3%)**

* NS ($p > 0.05$); ** NS ($p > 0.05$).

sex and type of surgery (Table 1). Table 2 gives the incidence of postdural puncture headache (PDPH) in the two groups according to age and sex. There was no significant difference between the groups with regard to the incidence of headache.

Figure 1 illustrates the incidence of postspinal headache in both groups combined, according to age and sex. Eight patients in each group received an epidural blood-patch. In Table 3, patients with postspinal, ordinary headache or no headache are compared according to age, sex, duration of headache, severity of headache and a pre-operative history of headache. Patients with postspinal headache were significantly younger than those with an ordinary headache, and the intensity of the headache was more severe and lasted significantly longer. A history of headache had no influence on the development of postspinal headache.

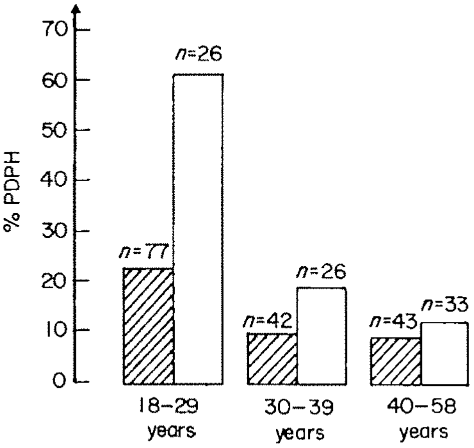

Fig. 1. The incidence of postdural puncture headache in the indomethacin and placebo groups combined, according to age and sex. ▨, Males; □, females.

Discussion

The overall incidence of postspinal headache in this series was 20.6%, with a mean age of 28.5 years. The incidence was 33% in patients less than 30 years old. Women were more prone to develop the headache than men in all age groups. Administration of indomethacin 100 mg rectally had no effect on the incidence of postspinal headache.

An ordinary headache occurred in 16.2% of patients following spinal anaesthesia. This headache was found to be distinctly different from the postdural puncture type; the patients were older and the headache was of shorter duration. There were more women in this group.

A history of headache was found in 17.4% of our patients pre-operatively. These patients suffered more frequently from headache of both types after surgery, but it did not seem to influence the occurrence of headache after spinal anaesthesia.

The mechanism behind the development of spinal headache is not entirely clear. One theory blames the dural puncture itself, which results in leakage of cerebrospinal fluid (CSF) through the tear in the dura. This is most pronounced in the first days after dural puncture, before the tear is sealed. CSF leakage hypotension will cause stretching of the cerebral meninges, especially in the erect position, and hence produces the classical picture of a low-pressure postspinal headache.[4,7] The other theory considers the origin of the headache to be psychogenic, in that if the patients expect to get headache, many of them will.[8] The study by Kaplan[9] supports this theory. He gave 100 young volunteers either a spinal puncture or a sham puncture in the lumbar region. All hundred were

Table 3. Patients compared with regard to postoperative headache and type of headache.

	No headache	Ordinary headache	PDPH
Males	121 } 156 (63.2%)	15 } 40 (16.2%)	26 } 51 (20.6%)
Females	35	25	25
Mean age (years)	33.8	38.5*	28.5*
Duration (days)	—	1.6**	3.9**
Intensity	—	4.0***	6.7***
History of headache	14 (9.9%)	17 (42.5%)	12 (23.5%)

* $p < 0.0005$; ** $p < 0.0005$; *** $p < 0.0005$.

told they had had a spinal puncture. An independent physician not knowing who had had a dural puncture did the follow-up and found that 22% developed a spinal headache in the sham group compared to 28% in the spinal puncture group.

Many prophylactic regimens against the headache have been advocated, but few have withstood controlled trials. The use of small-bore needles seems to be successful, with needles less than 26-G being reported to produce an incidence of headache of 0.69%.[10] Needles as small as 32-G have been used.[11] Clinical trials to compare two different sizes of spinal needles constantly find a reduced incidence of headache with use of the finest needle.[5,12,13] Use of prolonged recumbency after spinal anaesthesia was once advocated, but several studies have failed to show this to be of any benefit.[4,5,14] Use of the prone instead of the supine position in the postoperative period also failed to reduce the incidence.[15] Prophylactic bloodpatch and extra hydration have also been tried, but without success.[5]

Medicaments have also been tried prophylactically. The synthetic vasopressin analogue 1-desamino-8-D-arginine vasopressin is able to reduce the disability, but not the incidence of headache.[16] Kaukinen[5] found a reduction in the incidence of headache from 18% to 10% with the use of 100 mg indomethacin *per rectum* postoperatively. This was an open study and we have not been able to reproduce this in our double-blind study.

We have found a very high incidence of postspinal headache in spite of using 25-G needles. With this needle size, the reported incidence is usually low.[17] There are also reports of higher incidences.[3,18] Published reports vary regarding patient selection and study design. The mean age of our patients was only 35.5 years. This will result in a higher incidence as compared to studies with more elderly patients. Different quality of the follow-up may also be responsible and there may also be ethnical differences. All our patients were informed of the aim of this study. This may have sensitised them according to the psychogenic theory described earlier.

Our data demonstrate that ordinary headache after spinal anaesthesia is nearly as frequent as the typical postspinal headache. Such a headache could be provoked in many ways before, during and after surgery. This ordinary headache seems to be more benign than the spinal type in that it is of shorter duration and is less intense.

A pre-operative history of headache is regarded by some as a relative contraindication to the use of spinal anaesthesia.[5] Our data confirm that occurrence of both types of headache is more frequent in this group. We are, however, reluctant to use this as an exclusion criterion against spinal anaesthesia because of its low sensitivity in detecting patients prone to postspinal headache.

Conclusion

Postdural puncture headache in young adult hospitalised patients may be more frequent than previously reported. We found the overall frequency to be 20.6%. Prophylactic use of 100 mg indomethacin rectally had no significant effect on its frequency. Occurrence of ordinary headache is also frequent after spinal anaesthesia, but is less severe and of shorter duration. We now try to avoid spinal anaesthesia in males less than 30 years old and females under 45 years.

Acknowledgments

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SPECIAL COMMUNICATION

4th International Malignant Hyperpyrexia Workshop

Report of a meeting

Four years had passed since the last workshop in Banff, Canada but for those hoping for news of a breakthrough in our understanding of the biochemistry of malignant hyperpyrexia (MH), this meeting was to be a disappointment. However, there were a number of small pieces to fit into the puzzle. In MH, although primary calcium release is normal, calcium-induced release of calcium from the sarcoplasmic reticulum is abnormally great and can be increased by anaesthetic agents known to trigger MH. There appears to be a defect of the sarcoplastic reticulum membrane, which shows increased fluidity during exposure to halothane; dantrolene, but not amethocaine, inhibits this change. Studies on the erythrocyte of MH-sensitive (MHS) patients suggest that there is also a generalised membrane defect, in which membrane permeability is altered. During an MH crisis, even the erythrocyte membrane alters its properties.

In the writer's own field of interest, MH diagnosis and screening, there was a further slow but steady movement towards unity. It is probably fair to say that the only big event in diagnosis since the last workshop has been the formation of the European MH Group, largely due to the work of Dr F.R. Ellis (*Leeds*). This group was started in Sweden in 1983, and its pronouncements have influenced laboratories in the rest of the world. It is gratifying that all new laboratories are adopting the European MH Group's protocol and that many established ones are bringing their methods into line. However, a greater problem of MH diagnosis remains; the lack of high-quality normal control material. It seems, because of different countries' attitudes to health care and ethics, that only Scandinavia is going to produce this material, and until it has been considerably expanded, some uncertainty will remain in MH diagnosis.

In vitro testing is invasive (it requires an open muscle biopsy), so many workers are interested in finding the changes due to MH in other, more accessible tissues. Platelets are a favourite tissue to study, since they contain muscle elements. However, G.G. Harrison (*South Africa*) was unable to use the differences in adenine nucleotide profiles between normal and MH tissue as a diagnostic test. Lennmarken (*Linköping*) agreed, since his initially promising relaxation-rate studies, although they showed significant differences between normal and MHS subjects, could not be used in place of conventional *in vitro* testing.

A point of controversy at the meeting was the question of MH triggering by exercise and exposure to heat. Rutberg (*Linköping*) agreed with the European consensus that MHS subjects do not need to lead restricted lives. However, in the United States of America, MHS subjects are advised to avoid heavy work and exposure to heat, and are not allowed in the armed forces. In Japan another, for us previously unexpected difficulty arises: relatives of MHS subjects do not wish to be investigated, for fear it will reduce their chances of making a good marriage!

Masseter spasm is still an important reason for MH investigation; 50% of patients with masseter spasm as the single symptom prove to be MH positive. The masseter muscle contains a unique form of myosin, which may account for its unusual behaviour. When suxamethonium is given to an anaesthetised patient it *reduces* the gap by an average of 4 mm, whereas depolarising relaxants have no effect.

Mortier's contribution to the workshop was his unique experience of paediatric neurology: many patients suspected of being MHS in fact have some other muscular disease, but a number have MH *and* clinical muscle disease although this is unusual in adults. A Canadian paper put forward the view that coexisting clinical muscle disease does not invalidate *in vitro* testing.

The relationship between the neuroleptic malignant syndrome (NMS) and MH was the subject of several papers. NMS is a catatonic condition with high fever seen very occasionally after chronic ingestion of large doses of neuroleptics. It is still believed that the two conditions are separate entities, but the fact that both can be treated with dantrolene suggests some common aetiology.

The paper about nonanaesthetic triggers by Denborough was controversial: he believes there is a relationship between MH and the sudden infant death syndrome (SIDS). However, Ellis' group, with more stringent diagnostic criteria than Denborough's, have firmly refuted any connexion between SIDS and MH.

A final, almost humbling, contribution came from three ladies who represented MH patient organisations. They reminded the assembly that MH can be a tragedy for the family; great support and understanding are needed when an MH episode occurs, especially, as still happens, when it ends in death.

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Bowel sounds during intermittent positive pressure ventilation

Bowel sounds are a valuable clinical sign; the presence of normal bowel sounds indicates satisfactory intestinal function and their absence, an ileus. Normal bowel sounds are generated by the movement of intraluminal gas and fluid during peristalsis;[1] most of this gas is swallowed air and a small amount is formed by bacterial putrefaction.[2] Since both gas and fluid are necessary to produce bowel sounds, none will be

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heard in the absence of gastrointestinal air, in spite of normal peristalsis. Patients who receive controlled ventilation of their lungs with sedation, particularly with neuromuscular blocking agents in addition, swallow little air and in these patients the absence of bowel sounds may not indicate an ileus. Satisfactory gastrointestinal function without bowel sounds has been observed in a number of patients during controlled ventilation and one illustrative case is described.

A 26-year-old man was admitted to the intensive care unit following a road traffic accident. He had sustained multiple injuries which included a minor head injury, a penetrating eye injury, bilateral pneumothoraces and lung contusion, a ruptured spleen and fractures of the pelvis, humerus and femur. After laparotomy and splenectomy, he required controlled ventilation of his lungs and neuromuscular blocking agents were administered to assist ventilation and to avoid a sudden increase in intra-ocular pressure. He received continuous intravenous infusions of papaveretum and midazolam for analgesia and sedation. Enteral fluids were commenced 2 days after his laparotomy, *via* a nasogastric tube; full enteral feeding with Fortison Standard (Cow and Gate, Trowbridge) followed after 24 hours. No bowel sounds were heard but there was minimal aspiration of fluid from the nasogastric tube. The following day, his enteral feed continued to be absorbed but bowel sounds were absent and a chest X ray demonstrated the lack of gastrointestinal air (Fig. 1). The infusions of papaveretum and midazolam were discontinued and his trachea was extubated. His condition was unchanged the next day, apart from mild abdominal distension and the presence of normal bowel sounds. The development of gastrointestinal air was demonstrated on chest X ray (Fig. 2). He remained well and was discharged to the ward 3 days later.

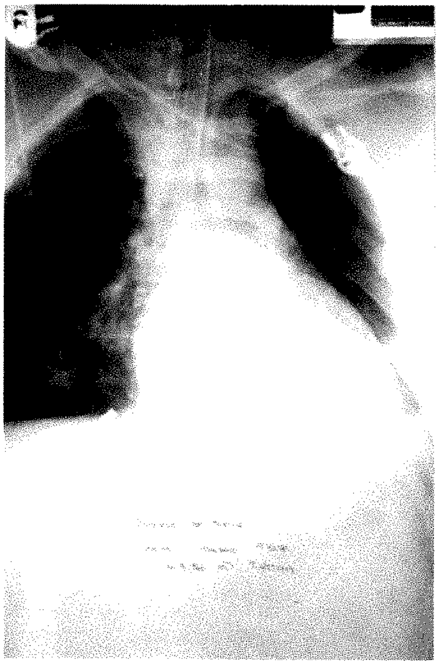
Fig. 1. Chest X ray before weaning from controlled ventilation (see text).

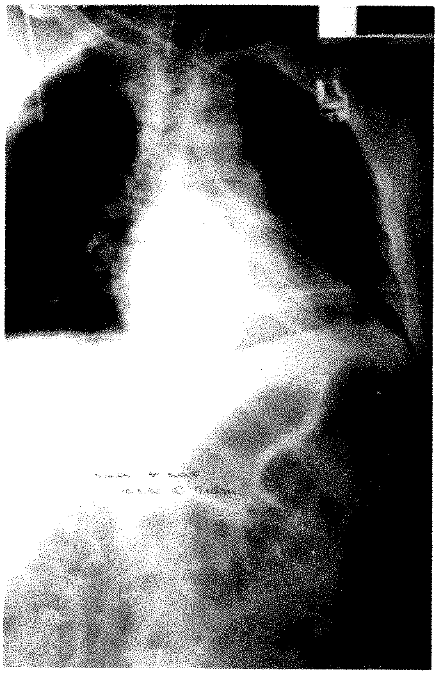
Fig. 2. Chest X ray after weaning from controlled ventilation (see text).

In patients who receive intensive care, many factors may influence gastrointestinal function, particularly surgery, disease,[3] sepsis, uraemia, anaemia and the administration of opioids.[4] Such patients also require adequate nutrition to aid their recovery and to avoid the consequences of malnutrition.[4,5] The assessment of gastrointestinal function in patients who receive intensive care, is difficult. The absence of bowel sounds may be interpreted as an ileus, and an indication for parenteral nutrition with its added complications and cost. In the absence of other factors, intestinal function generally returns 24–48 hours after intra-abdominal surgery, although it may recover at different rates in different parts of the gastrointestinal tract.[6] Patients who have controlled ventilation may not swallow sufficient air to generate bowel sounds and this sign is, therefore, unreliable as an index of gastrointestinal function in such patients. The simplest and most effective way to assess gastrointestinal function is by commencing enteral nutrition and measuring the volume of gastric aspirate.

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Alcohol vapour and the Normac analyser

It has been concluded that the infrared anaesthetic agent monitor Normac is compact, convenient and trouble-free for use in operating theatres.[1,2] One drawback of the Normac is that ethanol vapour in the sampled gas causes a spurious reading.[2]

We noticed an abnormally high reading immediately after tracheal intubation and after the use of a lignocaine spray (Fig. 1). Inquiry to the manufacturer revealed that, for the 8% lignocaine spray, 5.6 g of ethanol is included as a solvent in 80 g of spray solution. The package insert with lignocaine spray mentions nothing about any ethanol.

We should not therefore be misled by hidden alcohol when the Normac is in use.

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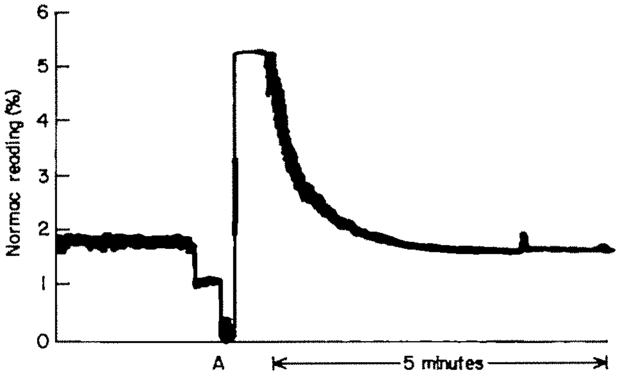

Fig. 1. Anaesthesia was induced with 2% halothane and the trachea intubated at point A following lignocaine spray. An abnormally high Normac reading was recorded.

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A difficult nasal intubation

An elderly lady was admitted for assessment prior to total glossectomy and hemi-mandibulectomy, with reconstruction, for a recurrent squamous cell carcinoma of the tongue.

Anaesthesia for an examination under anaesthesia consisted of etomidate, fentanyl and tracheal intubation after suxamethonium, and was provided by a senior anaesthetist; he reported that tracheal intubation (blind oral) was not a problem.

Her second anaesthetic was administered by a junior who was misled by the previous record and expected to find the situation unchanged; he administered thiopentone and, since controlled ventilation was possible, 7 mg of pancuronium! Laryngoscopy was unsuccessful and help was summoned.

Nasal intubation was required, but the irradiation induration and fibrosis of both the tongue and subcutaneous tissues made direct laryngoscopy impossible. Blind nasal intubation was quickly abandoned because of the danger of even moderate epistaxis.

Nasal intubation was achieved by guiding a tracheal tube into the larynx over a paediatric fibreoptic bronchoscope.

Since one nostril was obstructed by the bronchoscope and tube, a second Portex tube was placed in the other nostril, so that one-third of the gently inflated cuff remained outside; this was combined with apposition of the lips which enabled ventilation of the lungs to be maintained with ease. Thus laryngoscopy and bronchoscopy were performed without haste, the patient's gas exchange was maintained at all times and the dangers of removal and re-insertion of the bronchoscope and tube were avoided.

This case is reported firstly to emphasise the necessity of both keeping accurate records and reading those of others, and secondly, to bring to the attention of other anaesthetists a technique which may permit ventilation to be maintained during fibreoptic laryngoscopy.

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A.J. SANSOME

Arterial to end tidal CO_2 tension differences

The paper by Shankar *et al.* (*Anaesthesia* 1986; **41:** 698–702) is a useful contribution to our knowledge of gas exchange during controlled ventilation, but may I be permitted to comment on some errors in the discussion?

Regarding the third paragraph in the discussion, we showed[1] that *all* patients (except those with pulmonary embolus, carcinoid flush or hypovolaemia) had a positive slope to phase III of the single breath test for CO_2 (SBT-CO_2), that is, end tidal $P\mathrm{CO}_2$ ($P\mathrm{E'CO}_2$) is also peak $P\mathrm{CO}_2$. This is regardless of frequency or tidal volume. Many factors influence this slope; amongst these are tidal volume ($V\mathrm{T}$) and frequency (f), for when $V\mathrm{T}$ is large and f is small, the slope is least. Nevertheless, large volume, low frequency ventilation gives the greatest chance of $P\mathrm{E'CO}_2$ exceeding $Pa\mathrm{CO}_2$ in a well perfused, healthy lung, because gas distribution and mixing are better.

Alveolar $P\mathrm{CO}_2$ ($P\mathrm{ACO}_2$) varies cyclically, being least at end-inspiration and greatest at end-expiration. However, because of mixing in the heart and syringe, $Pa\mathrm{CO}_2$ sampled at the radial artery is the spatial and temporal mean of alveolar $P\mathrm{CO}_2$ (Riley's physiological integrator[2]) and therefore it is quite possible for $P\mathrm{E'CO}_2$ to exceed $Pa\mathrm{CO}_2$ thus sampled. This is not temporal deadspace; if it were, it would be a negative one! Temporal deadspace refers to the effect, during controlled ventilation, of ventilation ($\dot{V}$) being out of phase with perfusion ($\dot{Q}$). As inspiration proceeds, $\dot{Q}$ is reduced. Theoretically, temporal deadspace should be greatest during large $V\mathrm{T}$, low f ventilation, but under these conditions the alveolar deadspace fraction is least;[1] therefore we have argued[3] that temporal deadspace is of less importance than that caused by airway disease, that is, spatial, between- and within-units mismatching.

It is true that the reduced functional residual capacity (FRC) in pregnant patients should give rise to a greater cyclical variation in $P\mathrm{ACO}_2$, which increases the likelihood of sampling a $P\mathrm{E'CO}_2$ greater than $Pa\mathrm{CO}_2$. The lung model developed for controlled ventilation[3] supports this. However, the authors' statement that there is a greater incidence of positive phase III slope during Caesarean section anaesthesia, should be modified to read 'it is possible that phase III slope is increased . . . as a result of the reduced FRC.' The normal incidence of positive phase III slope is already 100%.

There is a second explanation for the zero and negative arterial to end tidal $P\mathrm{CO}_2$ differences. In obese patients with healthy lungs, there is a biphasic slope, reminiscent of phase IV of the N_2 closing volume test. The $P\mathrm{CO}_2$ of most of the alveolar gas is less than $Pa\mathrm{CO}_2$, but in the terminal part of the expirate, $P\mathrm{CO}_2$ rises rapidly and may exceed $Pa\mathrm{CO}_2$. Because pregnant patients have some features in common with obese ones, namely, reduced FRC due to a high diaphragm, and low total compliance,[4] they may also show this phenomenon, as discussed in my thesis.[5]

The important issues raised by the paper are firstly that gas exchange during Caesarean section with controlled ventilation would appear to be good. It may be comparable to that in children,[6] and is considerably better than in older patients, many of whom have airway disease and large alveolar deadspaces. The second point concerns the non-pregnant control group. These were only 2 years older than the pregnant patients, but their $Pa\mathrm{CO}_2 - P\mathrm{E'CO}_2$ is greater than that of our middle-aged patients.[1] This is strange, considering that they had never smoked and that deadspace is highly dependent on age.[1] The unexpectedly large deadspace that must cause this is, perhaps, a result of fairly vigorous (mean $Pa\mathrm{CO}_2$ 3.3 kPa) hyperventilation with 0.5–0.1% halothane. Perhaps this disturbs local pulmonary homeostatic reflexes. Our patients received nitrous oxide and opiates and were less hyperventilated (mean $Pa\mathrm{CO}_2$ 4.2 kPa).

Finally, the authors state that Raemer *et al.*[7] 'have shown several factors, namely changes in blood pressure, pulmonary blood flow, variations in compliance, anaesthetic agents, and surgical position lead to alterations in $\dot{V}/\dot{Q}$ ratios'. This is a misrepresentation.

Raemer *et al.* *speculated* on the effects of anaesthetic agents, surgical positioning, changes in temperature and pulmonary blood flow. Compliance was not mentioned. What they *demonstrated* was that for each of 11 variables, in about three of 15 patients (different patients for each variable), there was a significant correlation with $Pa\text{CO}_2 - P\bar{E}'\text{CO}_2$. This result is scarcely more significant than that which would be expected due to chance, and underlines the potential dangers of doing enormous batteries of linear regressions, in this case 165 of them. Raemer's data and conclusions do not support the statements made in the present paper.

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A reply

We thank you for allowing us to reply to the above correspondence. We appreciate Dr Fletcher's keen interest in our study and thank him for his expert comments on the various mechanisms operative during pregnancy to explain the negative or zero arterial to end tidal CO_2 tension difference. However, we would like to make the following points.

We agree with Dr Fletcher that Raemar *et al.* did speculate that a redistribution of the ratio of ventilation to perfusion throughout the lung would be expected owing to anaesthetic agents, surgical positioning, changes in temperature, changes in pulmonary blood flow, mechanical ventilation and cardiopulmonary bypass. The redistribution of ventilation relative to perfusion can produce changes in physiological deadspace that are reflected by changes in arterial to end tidal CO_2 tension. During Caesarean section anaesthesia, as was stated in our paper, one could expect there to be changes in pulmonary ventilation, pulmonary blood flow, blood pressure and pulmonary compliance. Therefore these changes are likely to influence the ratio of ventilation to perfusion and thereby arterial to end tidal CO_2 values. Hence we suggest that there is a wider scatter between $Pa\text{CO}_2$ and $P\bar{E}'\text{CO}_2$ during anaesthesia for Caesarean section.

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Nitrous oxide in early pregnancy

It is encouraging to note that for the fetus, general anaesthesia is as safe as regional analgesia when the mother undergoes cervical cerclage between weeks 8 and 28 of gestation. This observation still sheds no light on the period between 4 and 8 weeks of gestation during which the three germ layers give rise to tissues and organ systems in the human fetus. Prior to 4 weeks, abnormality of the blastocyst will probably cause no sign of pregnancy;[1] and after 8 weeks, changes occur mainly in fetal length, weight and cellular maturation. It is this period of between 4 and 8 weeks gestation, during which pregnancy is often only an unconfirmed suspicion in the mother's mind, that has been highlighted in animal studies.[2–4] These studies demonstrate fetal malformation in response to nitrous oxide exposure.

Extrapolation of animal studies to man is always contentious, but drug teratogenicity studies must always be performed in an animal and if a drug is found to have teratogenic properties in this model, it is doubtful whether it would gain a product licence for use in pregnancy. One therefore questions whether any stress and anxiety caused to the anaesthetist by administration of nitrous oxide during the fourth to eighth weeks of gestation would be wholly unjustified, but at the same time would not advocate administration of folinic acid to all women of child-bearing age!

The difficulty in performing any critique of the administration of nitrous oxide during this period is appreciated, and I look forward to the results of Drs Crawford and Lewis's second paper on the subject.

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Halothane and the Committee on Safety of Medicines

This letter is written at the request of our Division of Anaesthesia to express our considerable concern about the most recent circular (No. 18, September 1986) from the Committee on Safety of Medicines (CSM). This contains a section on halothane hepatotoxicity in which certain recommendations are made about the use of halothane. Most of these recommendations are sound and sensible but one states that the occurrence of unexplained pyrexia following exposure to halothane is an absolute contraindication to the future use of halothane for that patient.

This must surely be a very extreme view. Very many patients develop pyrexia following surgery, for a very wide variety of reasons, most of which are unexplained. To make the assumption that such episodes of pyrexia are due to the effects of halothane on the liver is unacceptable. We are urged by the CSM to take a careful anaesthetic history. This is highly to be commended but we dispute whether many patients will know, or care, whether or not they have had halothane in the past and whether or not this was followed by pyrexia. This might even be difficult to determine from the patient's previous casenotes if these happen to be available.

We feel strongly that the recommendations from the CSM regarding pyrexia following halothane anaesthesia should be withdrawn or modified. Otherwise, we foresee the use of halothane being abandoned as anaesthetists begin to practise defensive medicine. We will thus be deprived of the use of a most valuable agent.

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Children and explosive anaesthetics

The colleagues at Carshalton (*Anaesthesia* 1986; **41:** 1062–3) should not despair: cyclopropane is also much used 40 miles to the South! Their concern about current ignorance should not, however, be confined to the antistatic testing laid down by Hospital Technical Memoranda (HTM). Last year our engineers detected a slight decrease in the conductivity of a part of the floor of one of our theatres, caused at least partly by the introduction of a new floor-cleaning fluid. The theatre was closed and the floor was skimmed, which brought it back to an acceptable level of conductivity. Very proper, and a triumph for the HTM procedures!

A few weeks before that, an alert nurse had noticed that some children were coming from the ward wrapped in polyester blankets. The Divisional Supplies department had decided that the replacement of the former cotton ones would be an economy. As soon as our engineers demonstrated the unacceptable electrical conductivity of polyester we were assured a supply of cotton blankets. The supplies people were genuinely ignorant of the fact that clothing could constitute such a hazard.

Children frequently refuse to part with their favourite nylon nighties in exchange for our non-designer, but safe, hospital theatre garb: the problem is to persuade nurses to tell the anaesthetist of this! The great advantage of cyclopropane is that one may quickly anaesthetise a sleeping child, warmly wrapped up in his blanket, without the disturbance of inspecting his entire wardrobe. It is possible with other gaseous agents, but slower and possibly less pleasant. Will the long-awaited availability of sevoflurane solve our problem?

Meanwhile, vigilance and the education of all hospital staff about electrostatic precautions, must be the creed of those of us old-fashioned enough to use this valuable agent on children.

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P.A.D. Williams

Cyclopropane

This is a comment on the letter from the Queen Mary Hospital for Children, Carshalton (*Anaesthesia* 1986; **41:** 1062–3).

Cyclopropane is no longer in use at the Hospital for Sick Children, Toronto, where I have recently worked. Induction of anaesthesia with cyclopropane does not seem to be necessary, quite apart from the undesirable properties which this drug possesses. That hospital believes it to be always advisable to secure venous access and to start monitoring before the induction of anaesthesia. In the majority of children this is easily achieved and, of course, allows an intravenous induction technique to be used. The use of 27-G intermittent butterfly needles is almost painless. This approach rarely failed and over 85% of the children had no premedication.[1]

The economic problems associated with explosive anaesthetic agents and the necessary antistatic precautions needed are highlighted by Dr J.A. Lack in the same issue of *Anaesthesia* (p. 1063) as the letter from Carshalton. If cyclopropane ceased to be used there could be an economic benefit, with the relaxation of the present antistatic regulations. Surely the time has arrived when cyclopropane can be allowed to fade from the scene of anaesthesia with a wimper, and not a bang.

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Reference

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Manufactured drug dilutions for paediatric anaesthesia

The drug industry is often receptive to suggestions from anaesthetists in the presentation of marketed drugs. Paediatricians have many drugs at their disposal which are presented in dilutions suitable for the small doses used in paediatric medicine. However, paediatric anaesthetists in the United Kingdom are not so fortunate. In paediatric anaesthesia, drugs are often diluted for use from ampoules suitable primarily for adult dosage. This practice is not without hazard in terms of the calculations and accuracy of correct dilution[1] and the effectiveness of the diluted drug.[2]

In Canada, muscle relaxants and atropine are marketed in paediatric dilutions (for example, suxamethonium 20 mg/ml; d-tubocurarine 3 mg/ml; atropine 0.3 mg/ml) which I found to be a distinct advantage in the everyday practice of paediatric anaesthesia. The similar marketing of anaesthetic drugs in the United Kingdom in dilutions for children would represent another advance in British paediatric anaesthesia. Should representations be made to the drug industry?

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An inflatable paediatric wedge

It is sometimes difficult to position a child's head and neck for internal jugular cannulation and then for surgery. This problem can be solved by the use of an inflatable bag, similar to the one suggested by Carrie,[1] which is placed under the child's neck and shoulders.

The apparatus consists of an empty 1000-ml intravenous fluid bag connected by a drip extension tube to a three-way tap which is in turn connected to a sphygmomanometer bulb and valve. The bag may be placed under the neck and shoulders and inflated to extend the patient's neck to facilitate the insertion of an internal jugular line. It may then be deflated slightly in order to position the head and neck prior to surgery.

The bag also makes a suitable pillow when partly inflated and may be used as a bridge for thoracotomies in children, since it appears to be firmer and more stable than a rolled towel.

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Reference

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Pyrexia associated with a retroperitoneal tumour

A 37-year-old man presented with an acute abdomen after a blow to the epigastrium. A large retroperitoneal mass was found at emergency laparotomy, which extended from the body of the pancreas to the left adrenal gland. This was covered by fresh haematoma.

Anaesthesia was induced with a rapid sequence induction technique. Following preoxygenation, thiopentone 350 mg, suxamethonium 100 mg and fentanyl 200 mg were given and anaesthesia was maintained with oxygen 30%, nitrous oxide 70% and halothane 0.5%. Muscle relaxation was continued with atracurium 40 mg.

Anaesthesia after induction was complicated by persistent elevation of systolic blood pressure compared with pre-operative values (rising from 130 to 220 mmHg), a sinus tachycardia (110 beats/minute rising to 160 beats/minute), an increased end tidal $P\text{CO}_2$ of 7.3 kPa despite a minute volume of 10 litres, an increasing core temperature (35.5°C rising to 37.5°C) and peripheral vasodilatation. The surgeons also remarked on the warmth of the peritoneal cavity. There was, however, no muscle rigidity.

These changes persisted throughout the opening of the anterior abdominal wall and were exacerbated by handling the tumour mass, despite a blood loss greater than 1 litre. Active measures were therefore taken to cool the patient. The use of halothane was stopped and the breathing system was changed to one which was vapour-free. No dantrolene was given and no evidence of intra-abdominal sepsis was found.

Once the haematoma had been evacuated and handling of the tumour had ceased, the hypertension, tachycardia and pyrexia rapidly settled. It was not therefore thought necessary to screen the patient for susceptibility to malignant hyperpyrexia.

On histological examination, the tumour was found to be an alveolar soft-part sarcoma. This is a very rare tumour, accounting for only 1% of all sarcomata[1] and tends to arise from the deep tissues of the extremities, typically from within skeletal muscle such as the muscles of the thigh.[1,2] They are usually fatal. Such tumours were previously defined as malignant non-chromaffin paraganglionomata.[1,2]

Have other anaesthetists experienced such marked physiological changes, mimicking malignant hyperpyrexia, during anaesthesia for patients with large retroperitoneal tumours?

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Prolonged neuromuscular blockade in a patient with a normal genotype

The case report by Sapsford and Bush (*Anaesthesia* 1986; **41:** 844–6) is interesting. The postulate of a different, yet to be described, genotype responsible for the prolonged apnoea in their patient is a theoretical possibility. Muscular paralysis lasting approximately 140 minutes with significant fade in the train-of-four response, makes me wonder whether a non-depolarising muscle relaxant such as pancuronium, with a clinical duration of action of 120–150 minutes, near administered accidentally instead of suxamethonium.[1] This would have been suspected if a neuromuscular blockade monitor was used from the time of administration of the muscle relaxant.

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Reference

1. Miller RD, Saverese JJ. Pharmacology of muscle relaxants and their antagonists. In: Miller RD, ed. *Anesthesia.* New York: Churchill Livingstone, 1986: 889–943.

A reply

Thank you for the opportunity to reply to this letter. We entirely agree that the response to neuromuscular stimulation as described in our case report could have been produced by a non-depolarising muscle relaxant. These characteristics are also shown by patients recovering from a prolonged neuromuscular block following only suxamethonium administration.[1] However, we think Dr Cherala's suggestion to be unlikely for the following reasons, although we accept that accidental administration of the wrong drug can unfortunately happen and should always be considered when an unexpected response occurs.

The dose of suxamethonium administered to our patient was 75 mg, equivalent to 1.5 ml of solution. If this had been pancuronium then 1.5 ml of solution would have contained 3 mg pancuronium. In a 51-kg subject this is equivalent to a dose of 0.058 mg/kg. This is between 40% and 60% of normally recommended dosages for pancuronium for intubation (0.1–0.15 mg/kg). Our recommended dose is 0.12 mg/kg.[2]

The onset of action of pancuronium differs from suxamethonium; the latter achieves profound paralysis within 60 seconds and produces excellent conditions for intubation as in our patient, whereas tracheal intubation after pancuronium in a dose of 0.058 mg/kg may necessitate a wait considerably in excess of 2 minutes (see Table 1). The duration of pancuronium 0.058 mg/kg is unlikely to be 140 minutes. A dose of 0.13 mg/kg of pancuronium has a duration of action of 41 minutes,[2] whilst Bennett and Daughety[3] showed

Table 1.[4]

Dose (mg/kg)	Mean time to peak effect (minutes)	Mean duration (minutes)
0.050	7.0	20
0.080	2.5	40
0.100	2	60

the mean duration of 0.15 mg/kg and 0.1 mg/kg to be 53 and 31 minutes, respectively. Our patient had no detectable response to neuromuscular stimulation for 105 minutes.

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Broken control on a Selectatec vaporizer

A potentially hazardous fault was found recently on an Enfluratec 3 vaporizer. At the pre-use check, the vaporizer control knob could be turned clockwise beyond the 'off' position (Fig. 1). Examination of the knob showed the plastic cam beneath had been fractured, by being turned off forcefully (Fig. 2). No vapour was delivered in this position, but the fragment could have jammed the control knob at any setting. Fortunately, this was a Selectatec vaporizer, which could be removed from the anaesthetic machine at once. This has occurred before, when it also indicated a faulty, potentially hazardous, control knob cam.

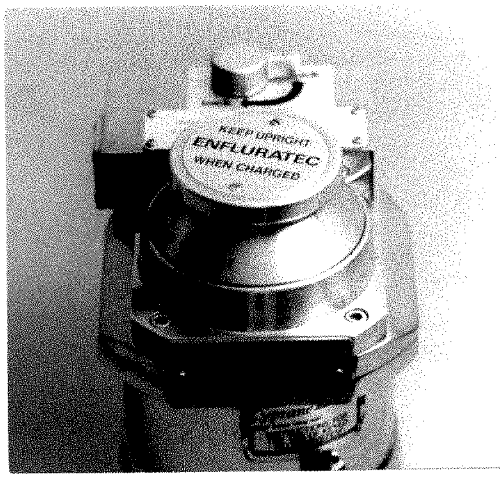

Fig. 1. Vaporizer control knob, turned beyond the 'off' position.

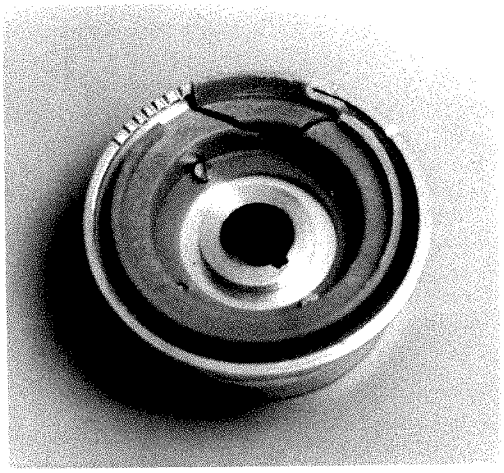

Fig. 2. Underside of control knob, with fractured cam.

The pre-use check of anaesthetic machines should include a full range check of the vaporizer control knobs, to exclude an excessive range of movement. That range usually does not fall exactly within the marked limits, so it would be useful if the manufacturers were to indicate the permitted tolerances.

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Postscript

The vaporizer was donated as part of an agreement on the supply of enflurane and not purchased in the usual way, so it was not listed as part of this Department's equipment and was not serviced. This incident has led to a revision of equipment registration and arrangements for maintenance, which other departments may wish to copy.

A reply

Thank you for the opportunity of replying to this letter. The vaporizer was supplied in April 1981 and our records show that it has not been serviced since that date.

Ohmeda has a policy of continued development and updating of current products; a redesign of the mechanism in question took place a little while after this particular vaporizer was sold.

Had routine maintenance been carried out in accordance with the recommendations of the manufacturer (and the Department of Health and Social Security), we believe that this occurrence would not have taken place.

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B. SMITH

Minitracheostomy—a modified procedure

The insertion of a minitracheostomy tube into the trachea of a patient who is breathing spontaneously, is sometimes made more difficult by the respiratory excursions of the cricothyroid membrane under the skin and subcutaneous tissues. This relative movement between skin and membrane results in the track created by the scalpel blade being lost while attempts are made to insert the stillette.

The simple manoeuvre of insertion of a 20-G needle percutaneously into the trachea at the level of the first tracheal ring on either side, fixes the skin relative to the membrane and facilitates passage of the stillette and minitracheostomy tube; the needle is removed once the stillette is inserted.

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B.A. Willis

Minitracheostomy

The timely appraisal of minitracheostomy (*Anaesthesia* 1986; **41:** 931–5) emphasises the value of this procedure in a variety of clinical problems which the authors have encountered. Complications are few but can be serious, as the two following case histories illustrate.

A 36-year-old man with cor pulmonale related to long-standing bronchiectasis that had necessitated pneumonectomy, was admitted in acute respiratory failure. Eleven days of controlled ventilation of the lungs were required before respiratory independence could be achieved but sputum expectoration remained a problem. A minitracheostomy was easily performed and some bleeding occurred (which is common). It rapidly became obvious that the airway was obstructed. The trachea was re-intubated and a large amount of blood removed from the trachea. No further treatment was needed. The patient eventually had a tracheostomy and negative pressure ventilation but died before discharge from hospital.

A 61-year-old man was admitted in respiratory failure with a 30-year history of bronchiectasis. He required controlled ventilation of the lungs for 12 days but his airways rapidly filled up with tenacious secretions if the trachea was extubated. A minitracheostomy tube was easily inserted. Bilateral surgical emphysema of the neck and face developed 2 hours later; this progressively worsened and re-intubation was required. The problem then resolved and the patient made an uneventful recovery and eventually returned home.

There is no doubt in our experience that this procedure is helpful in postoperative patients and some ITU problems. We are less convinced of its role in handling chronic lung disease, which tends to be intractable to any therapy. These cases illustrate that serious problems do occur, albeit infrequently. Continued evaluation and wide clinical usage will enable this procedure to gain a fuller appraisal in the course of time.

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A. Wagstaff
R. Sparling
D.W. Ryan

Transvenous misplacement and loop formation of spring guide wire

Central venous catheters are used increasingly for monitoring and therapeutic purposes. This is a report on a complication during insertion, not reported before.

An adult male with a cancer of the testicle, previously treated by orchidectomy, chemotherapy and radiotherapy for spinal metastases (C_7–T_3), was admitted to our department for central venous catheterisation before the sixth course of chemotherapy.

Puncture of the right subclavian vein was performed but during introduction of a spring guide wire a little resistance was felt; this disappeared on slight pressure. The wire was introduced for another 5 cm, the needle withdrawn and the catheter mounted over the wire. However, the catheter could only be inserted 1 cm. It was decided to remove the catheter and wire. The catheter came easily, but the wire was stuck. An X ray (Fig. 1) showed that the wire had formed a loop and was tied with a knot. The wire was removed surgically. Operation showed that it had passed into a nearly occluded subclavian vein, through the posterior wall into the anterior scalenus muscle. The postoperative period was uneventful.

Prolonged central venous catheterisation has been shown to cause a high frequency of thrombosis.[1,2] The material of the catheter is important[3] and other factors include the pH and composition of infused solutions, the presence of infection and the technique of venepuncture. Histological examination of thrombosis showed myxomatous degeneration within the thrombus.

The patient had five central venous catheters of unknown composition inserted in the right subclavian vein within 6 months for infusions that contained cytotoxic drugs, antibiotics, electrolytes and mannitol. One catheter was removed because of infection. A radiation reaction with laryngeal discomfort had occurred 3 months previously.

The resistance during insertion of the guide wire was

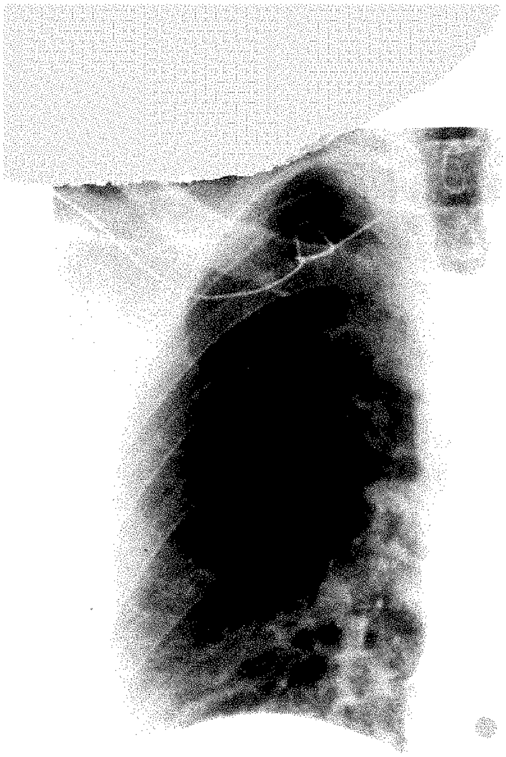

Fig. 1.

similar to that often felt when the wire emerges from the bevel of a needle close to the internal vessel wall. The occlusion and reorganising phenomena in the vessel might explain the ease with which the soft end of the wire pierced the vessel. Once through the vein, the wire passed into the soft scalenus anterior muscle. When the wire met the posterior fibrous membrane of the muscle, it changed course anteriorly to make a loop and a knot.

Patients with tumours sensitive to chemotherapy often require repeated central venous catheterisation. An increasing deterioration of the condition of the veins should be anticipated. Catheters of nonthrombogenic material should be used and a regular change between veins should be considered in order to reduce complications.

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Pleural aspiration with a central venous catheter

The Secalon T over-needle central venous catheter (British Viggo, Swindon, England) can be used to drain a pleural effusion since it combines the advantages of conventional techniques for this procedure. It includes a simple on/off device, the FloSwitch, which may be used to occlude the cannula as the trocar needle is withdrawn.

A 73-year-old, spontaneously breathing, patient presented with multiple pathology; the chest X ray showed a large right pleural effusion and his respiratory rate was 40 breaths/minute. Management was to include drainage of the effusion, and laparotomy was considered.

Conventional methods of pleural fluid aspiration when formal chest drainage is not indicated, require either a metal needle or a cannula-over-needle technique. A metal needle can cause trauma to the parietal pleura and carries the risk of pneumothorax;[1] if positive pressure ventilation of the lungs is then started, tension pneumothorax may develop. The use of a cannula inevitably involves a disconnexion to atmosphere when the needle is removed, and this entails the risk of aspiration of air into the pleural space,[1] especially in patients with marked inspiratory effort and tachypnoea. The Secalon T might help to overcome these potential problems.

Consequently, following the usual preparations, a 16-gauge 130-mm Secalon T cannula attached to a syringe was introduced through the chest wall and the effusion located by gentle aspiration with the syringe. The needle was immediately withdrawn, at the same time closing the FloSwitch; the cannula was then advanced into the effusion. The drainage system was attached without the need to hurry, the FloSwitch was opened and the effusion aspirated slowly to dryness; 1850 ml of transudate was collected. An immediate improvement in the patient's condition was noted and the cannula was then removed. A chest X ray confirmed success.

Although I am unaware of previous reports, the manufacturers have informed me that this cannula, which is available in a range of sizes, has been used successfully in this way on several occasions. Thus, it would seem sensible to consider it as a useful addition to the equipment available for therapeutic and diagnostic pleural aspiration.

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Reference

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Primary amyloidosis and skin damage

The protection of the eyes of unconscious patients is an important part of the anaesthetist's role: this is usually done either by placing adhesive tape over the patient's eyes or by using antibiotic ointment. This is a report of an unusual, but important, complication of the use of adhesive tape in a patient who had primary amyloidosis.

The patient was a 47-year-old man whose condition of primary amyloidosis was diagnosed 2 years previously. He complained of a recurrent spontaneous petechial rash around the eyes, and the diagnosis was subsequently confirmed by rectal biopsy. He had been treated since that time with intermittent courses of melphalan and prednisolone. He was scheduled to have a left thoracoscopy with pleural biopsy because of a pleural effusion associated with a 7-month history of increasing shortness of breath. Pre-operatively a purpuric rash was noted over his anterior chest wall; the platelet count and clotting screen were both normal.

His eyes were covered during the operation with Blenderm adhesive tape and, in addition, an ECG electrode was placed on his forehead. When the tape was removed from his left eye at the end of the procedure a small piece of skin from the upper eyelid (approximately 0.5 × 1.0 cm) was detached with the tape and there was bleeding associated with an extensive ecchymosis around the site where the tape had been placed. The protective cover over the right eye and the ECG electrode on his forehead were then both removed with great care but, despite this caution, extensive haemorrhagic rashes appeared at both of these sites and there was also some frank bleeding. The site of the ECG electrodes on the chest wall also showed purpura, though these were much less marked than those around the eyes.

He was treated with Polyfax ointment to the eyes and the periorbital ecchymoses subsequently resolved with time. The rash, however, caused the patient and his family some distress.

Amyloidosis is classified into primary and secondary, and the manifestations of the disease depend very much upon where the amyloid is deposited. This patient had both renal and cardiac involvement but his main symptoms were related to the skin; he was prone to develop spontaneous bruising around the eyes and he also had gross deposition of amyloid in the subcutaneous tissues of his fingers and hands.

There are few case reports of patients with amyloidosis who undergo anaesthesia and surgery. Welch[1] described a case of a man with primary amyloidosis who developed resistant cardiac failure after a minor surgical procedure; in this report he mentioned the high incidence of skin involvement (40%) and the fact that patients with amyloidosis may develop purpura either spontaneously or with minimal trauma secondary to amyloid deposits in the blood vessels. These patients are also prone to develop bullous eruptions owing to intradermal deposition of amyloid.

Other reports[2] have underlined the fact that focal or generalised haemorrhages are commonly encountered clinical problems in these patients despite the absence of abnormalities in clotting tests.

This case is reported in order to emphasise the extreme fragility of the skin in patients who have skin involvement with amyloidosis; great care must be taken to ensure that trauma is minimised in the peri-operative period. The use of protective eye tape may actually cause harm.

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Bristol

J. Dixon

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Epidural meptazinol

The letter by Drs Francis and Lockhart (*Anaesthesia* 1986; **41:** 88–9) which describes their experience with epidural meptazinol has only just come to our attention. The problems of pruritus, nausea, vomiting and hypertension which they encountered are somewhat surprising in view of the favourable experience which we reported[1] and that of Budd *et al.*[2]

Differences between our patients and theirs may account for the discrepancy. Their patients were all post partum and may have had opiates during labour or delivery. Pregnant women and women immediately after delivery are prone to vomiting, dizziness and sweating due to a variety of obstetric complications and administered drugs. No mention is made of any of these possible factors in their report. All our patients had had major gynaecological surgery. None received any opiates either before or during surgery. Our conclusions were that meptazinol gave good quality pain relief which lasted for only 2 hours. We agree with the comments made more recently[3] that the major problem with epidural meptazinol is short duration of action.

We have since given single doses of 60 mg (blind) to a further 30 patients after major gynaecological surgery, and continuous epidural infusion to seven patients after major upper abdominal surgery at a rate of

30 mg/hour for 60–64 hours. On no occasion have we encountered the problems described by Francis and Lockhart.

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I.T. Campbell
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D.M. Catley

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Disconnexion hazard

There is a hazard associated with the use of the Hewlett–Packard Capnometer Airway Adapter, but there is also a simple but effective solution to the problem.

The airway adapter consists of an aluminium body with push-on polypropylene couplers for attachment between the catheter mount and the ventilator tubing. These couplers are disposable; however, they are often re-used, with the result that the metal to plastic junction becomes loose and liable to disconnexion.

These couplers may be wired in place with stainless-steel wire of the type used for sternotomy sutures to avoid such potentially disastrous occurrences. Wiring is best carried out in the manner shown in Fig. 1, with the twisted ends of the wire being at the female end to avoid fouling of the connexion.

It is recommended that wiring is carried out in addition to regular inspection and replacement of the polypropylene connectors until a locking device is introduced by the manufacturer.

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London W2

P.L. Goldberg

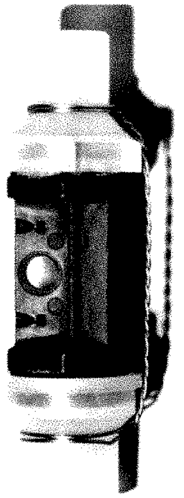

Fig.1.

Leasing of equipment

Limited anaesthetic departmental budgets and the high cost of anaesthetic equipment and machines are of common concern. Our home industry complains that British anaesthetists use equipment that is old, antiquated and, by inference, less safe. This results in less production and higher unit costs of new equipment. Other industries have faced and solved similar problems to their mutual benefit, and their solution involves the lease of equipment from the manufacturer.

There are currently a number of independent anaesthetic equipment manufacturers, and competition for lease contracts would keep costs at a reasonable level. This would present a relatively constant annual bill to the anaesthetic budget for modern, and readily updated equipment. Industry would have a predictable, stable market, and a basis to develop a universal standard for machines.

A range of machines and ventilators is necessary in each department to assist with teaching and development. The lease of equipment need not limit this practice. Industry might be prepared to buy back, or give an allowance for, old and obsolete machines. This would enable meagre hospital budgets to recoup funds for other desperately needed projects.

The concept of leasing medical equipment is not new, but in the current financial climate it may solve some of our problems and perhaps should be given further serious consideration.

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T.D. Wauchob

Unwanted equipment

We write with regard to the subject of unwanted equipment that is present in probably every anaesthetic department in the United Kingdom.

What is no longer required by one department could profitably be used by another. However, there are two problems involved: firstly, a means of publishing, within our professional circle, what items various departments have available; and, what others want. It is possible that a NHS publication to this end is periodically available, but we are not aware of its existence. The second problem is that of payment: but this is not insurmountable.

We wish to acquire a Manley Servovent and a BOC carbon dioxide absorber unit: we have recently discovered, during a recent idle social conversation with a consultant anaesthetist's relative, that his department in the North East of England may have a Servovent ventilator and an Engström CO_2 analyser monitor available for resale!

Do readers think that there might be sufficient equipment lying fallow, to warrant actively trying to correct the situation?

Welsh Plastic Reconstructive Surgery and Burns Centre,
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E.R. Emmanuel
D.J. Dye

To chew or not to chew

On removal of an oropharyngeal airway from a patient with a limited English vocabulary, some chewing gum was found to be adherent to its distal end. There was a 25% reduction in the diameter of the airway which, however, did not cause respiratory obstruction.

Should we ask patients if any food or chewing gum has passed their lips during the previous 6 hours? Are sucking sweets and chewing gum considered by patients as eating food? One suspects not.

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Edgware,
Middlesex HA8 0AD

R.J. Cohen

Difficulty in extubation

There appears to be a potential hazard associated with the use of preformed RAE tracheal tubes. A 7-year-old child was recently anaesthetised for an adenoidectomy. An oral, uncuffed size 5.5 (internal diameter) RAE tube was chosen and proved to be a good fit. A Boyle–Davis gag with a size 3½ Doughty split blade was inserted to achieve surgical access.

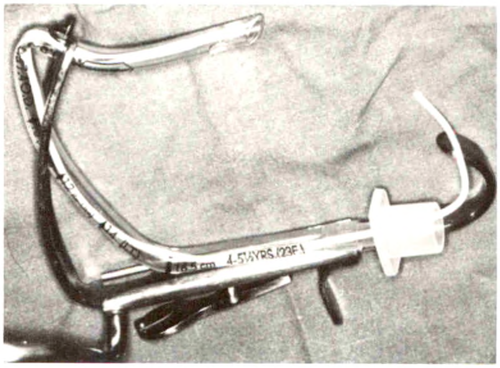

Fig. 1.

Anaesthesia was uneventful but at the conclusion of surgery the surgeon was unable to remove the blade without removal of the tube. Figure 1 shows the reason for this; the tracheal tube had slipped into the blade of the gag and its posterior movement became limited by the retaining ring, around which it had then began to kink (the stylet has been inserted to demonstrate the amount of kinking possible). The wedged tube was removed only with difficulty, and later attempts to push an unused tube of the same size through the blade required considerable force.

It is not clear how this incident occurred but one possible explanation is that the tube had become softer with the higher temperature in the mouth and so could be pushed into the groove. This observation serves as a reminder that the sizes of the tracheal tube and blade should be carefully matched, because the tube used was the recommended size for a 4- or 5-year-old.

The Queen Victoria Hospital,
East Grinstead,
Sussex RH19 3DZ

P. Wood

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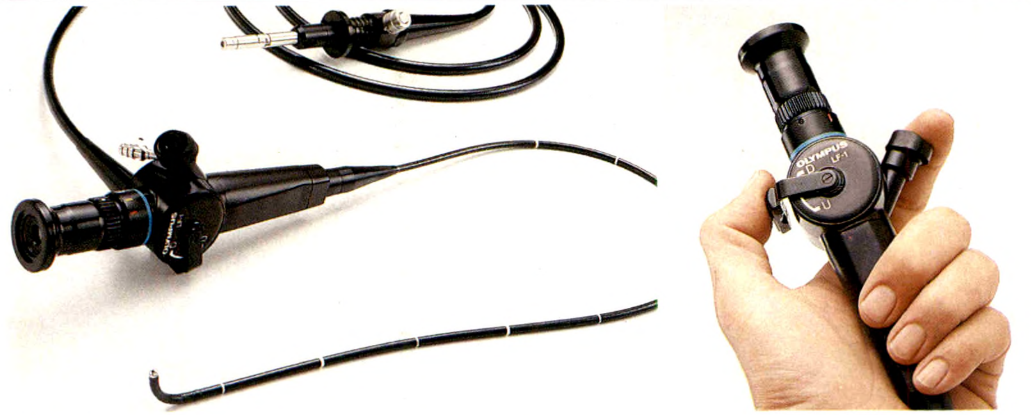
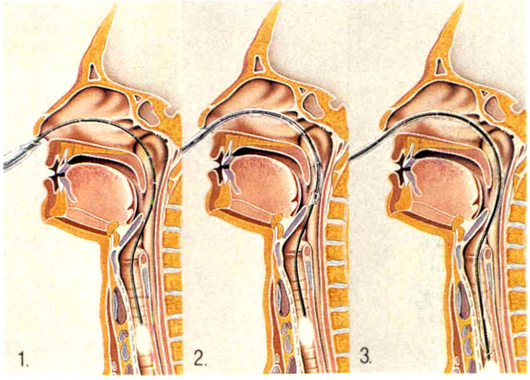
LF-1 INTUBATING FIBERSCOPE

Specifically designed for anaesthetic use



Fiberoptic bronchoscopes have proved their value for intubating difficult cases*. The LF-1, which is less expensive than current models of bronchoscopes, has been designed to meet the special demands of the procedure. The slimline insertion tube allows a wide range of ET tube diameters to be used, whilst the ergonomic design makes it simple to operate. The LF-1 is an instrument from the OES (Olympus Endoscopy System) range, with the proven benefits of increased durability and immersibility for sterilisation.



1. The LF-1 with the endotracheal tube near the control body, is passed transnasally deep into the trachea.
2. The ET tube is advanced through the nasopharynx using the LF-1 as a 'guide wire'.
3. The ET tube is placed in the trachea, under direct visual control.

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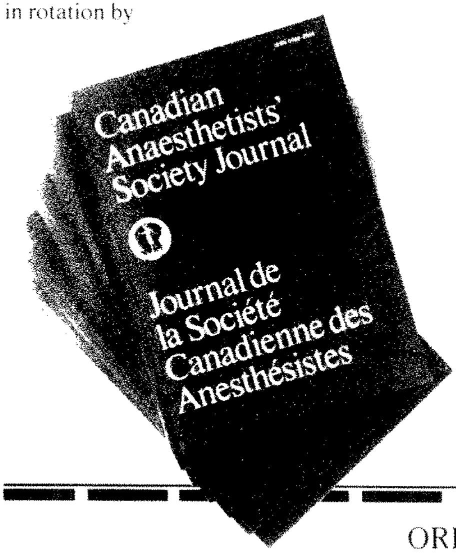
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The collator of this section is Dr L. Kaufman, MD, FFARCS, Anaesthetic Office, The Rayne Institute, University College, University Street, London WC1E 6JJ. Dr Kaufman is prepared to provide on request, and at a modest charge, a new additional service to our readers. References from January 1984 have been entered on data base. The data are held on Dbase II, and are available on disk, together with a program providing search facilities. Enquiries direct to Dr Kaufman at the above address please.

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Anaesthesia, 1987, Volume 42, page 229

Stop Press

Ivan Whiteside Magill

Sir Ivan died on 25th November, 1986. A full Obituary will appear in a later issue of the journal.

Anaesthesia in Older Age Groups

An exploratory meeting will be held on Thursday, 26 March 1987 at the Haworth Building of the University of Birmingham.
Information: Dr W. G. B. Clark, Good Hope General Hospital, Sutton Coldfield, West Midlands B75 7RR.

REGIONAL ANESTHESIA:

Techniques and Clinical Applications

HAROLD CARRON,
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JOHN C ROWLINSON

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This work is designed as a practical reference for all physicians and other professionals who use regional anaesthesia in their practice. Written by well-known teachers in the field, the text provides clearly defined guidelines on anatomical considerations, concise descriptions of surface landmarks and techniques for performing regional anaesthetic procedures.

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MUSCLE RELAXANTS:

Basic Clinical Aspects

Edited by RONALD M KATZ

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This new pocket-sized guide provides a quick and thorough reference to the clinical uses of muscle relaxants.

Its timely coverage includes the physiology and pharmacology of neuromuscular transmission, and the clinical use of such muscle relaxants as atracurium and vecuronium. Muscle relaxant antagonists, complications and controversies are discussed in detail. The use of muscle relaxants in patients with heart disease, renal failure and children are also included.

A PRACTICE OF ANESTHESIA FOR INFANTS AND CHILDREN

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This book is a ready reference for the practical management of anaesthesia for paediatric patients. It provides more than the limited direction of a manual by emphasising the principles and psychology upon which the practice is supported.

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The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36**: 119–25.

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BOOKS AND OTHER MONOGRAPHS

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Rhodes AJ, Van Rooyen CE, *comps. Textbook of virology: for students and practitioners of medicine and other health sciences,* 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

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Agency publication

National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968—June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series* **10**: Data from the National Health Survey, No. 69) [DHEW publication No. (HSM) 72–1036].

OTHER ARTICLES

Newspaper article

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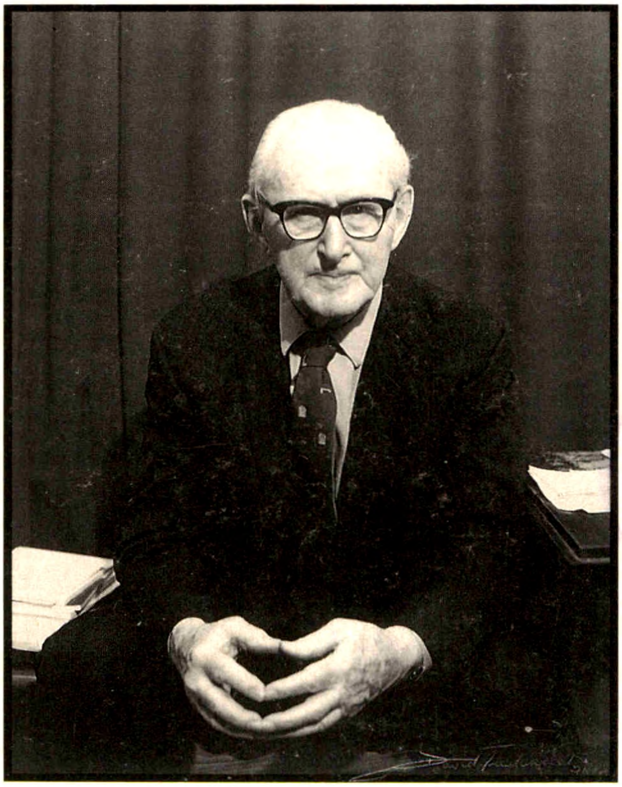

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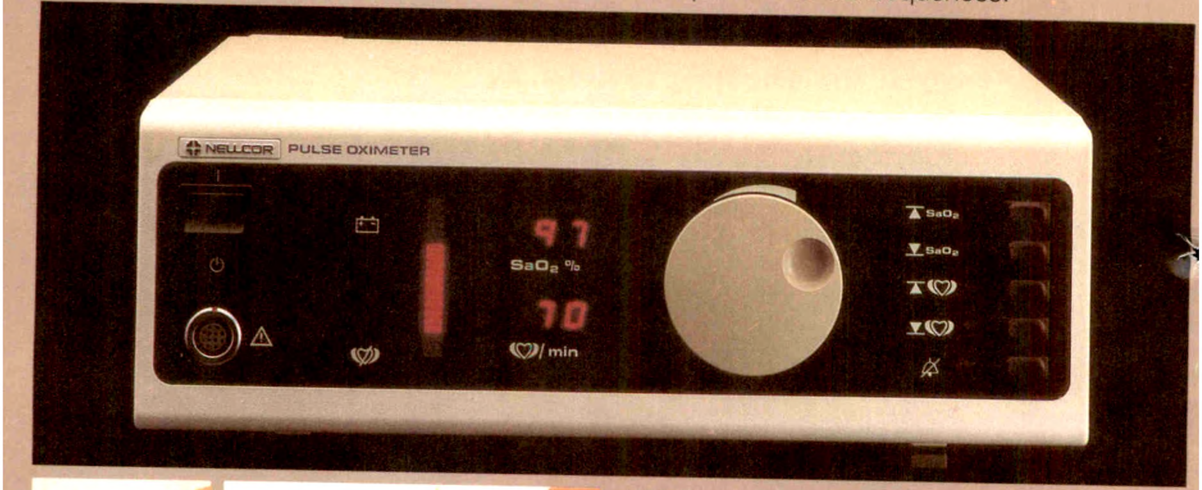

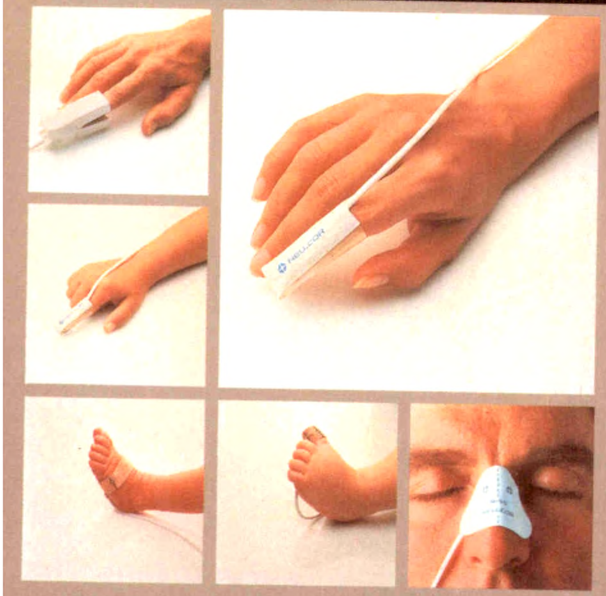

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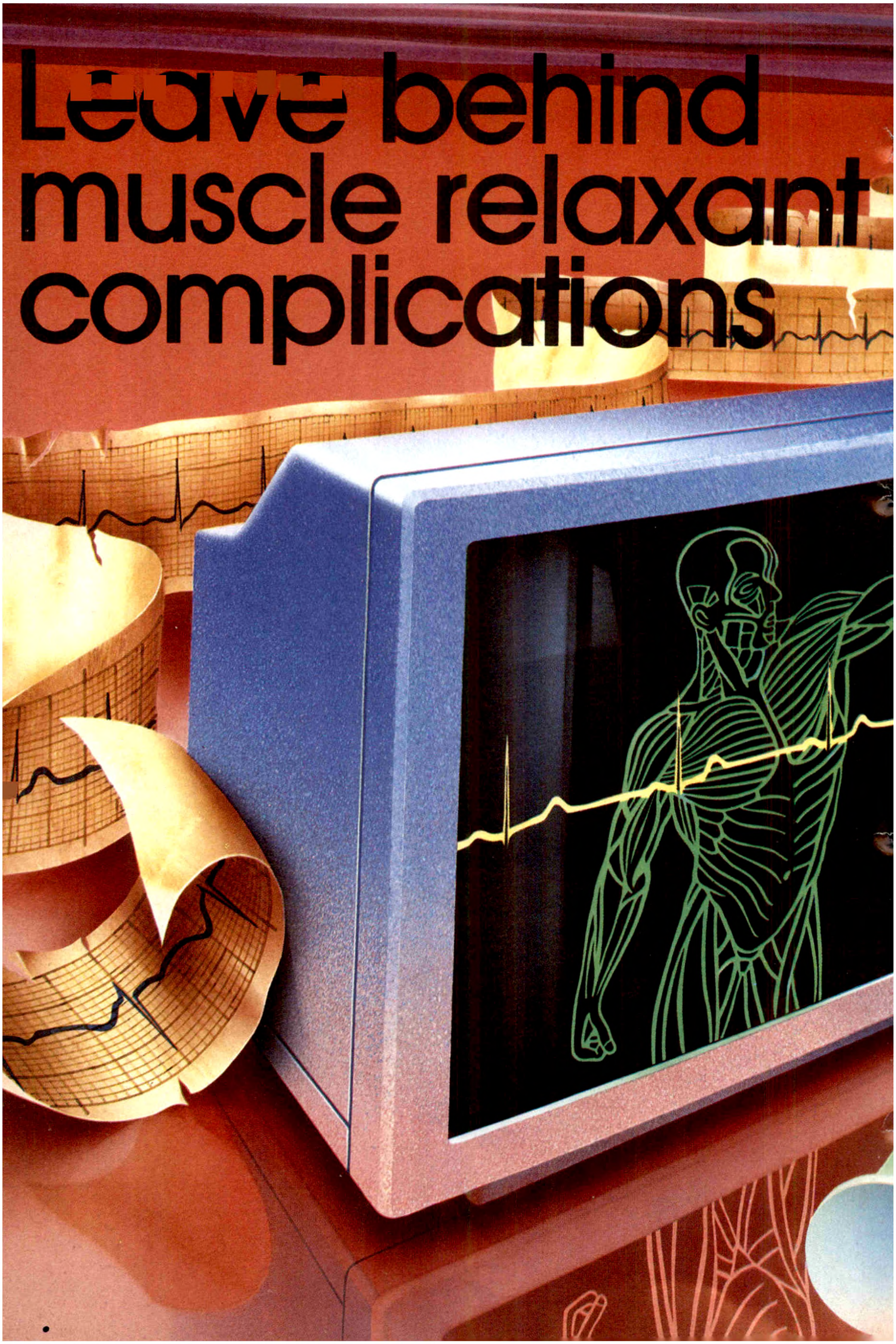
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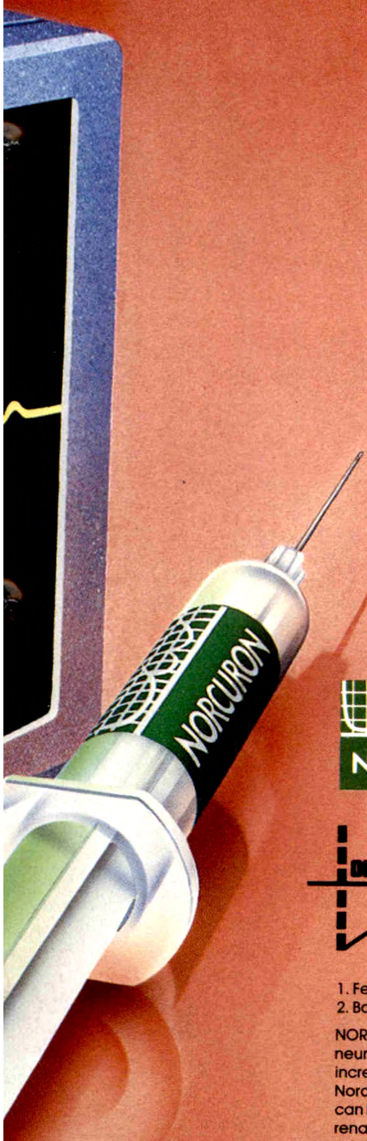

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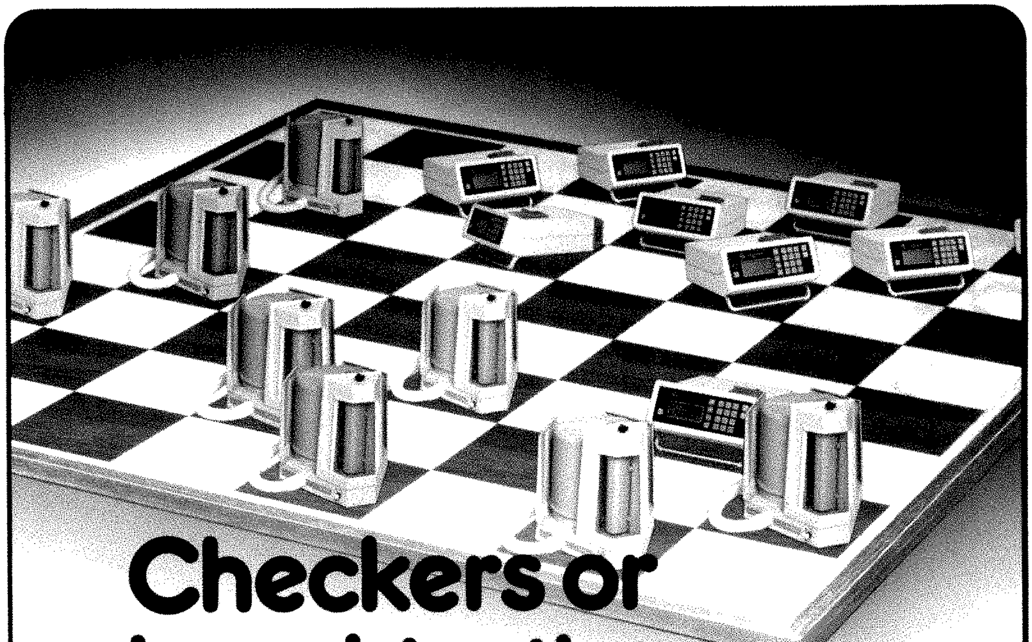
1. Feldman SA; Clinical Experiences with Norcuron, Excerpta Medica (1983) 199-200
2. Basta SA; Clinical Experiences with Norcuron, Excerpta Medica (1983) 183-184

NORCURON ampoules of 4mg vecuronium bromide with 1ml ampoule water for injection. **Uses:** non-depolarising neuromuscular blocking agent of short to medium duration. **Dosage:** intravenous initial 0.08 to 0.10mg/kg incremental 0.03 to 0.05mg/kg. **Contra-indications:** none known. Since there is no experience with the use of Norcuron in pregnant women, it cannot be recommended during pregnancy. Clinical studies show that Norcuron can be used in childbirth by Caesarian section without effect on the newly born child. **Precautions and warnings:** in renal insufficiency a slight prolongation of neuromuscular block can be expected. Use very small doses, and extreme caution in myasthenia gravis or myasthenic syndrome unless prolonged post-operative respiratory assistance is intended. Dose carefully in myopathy, severe obesity, electrolyte disturbances, altered pH and after poliomyelitis or dehydration. Neuromuscular blockade can be reversed with adequate doses of neostigmine together with atropine. **Interactions:** it is dangerous to give depolarising drugs (e.g. suxamethonium chloride) following a non-depolarising drug (e.g. Norcuron). Alkylating drugs (nitrogen mustards) may be a hazard in anaesthesia involving muscle relaxants. Anaesthetics, other drugs and the condition of the patient, may affect the magnitude and or duration of action of NORCURON – see Data Sheet. **Side-effects:** none during clinical testing.



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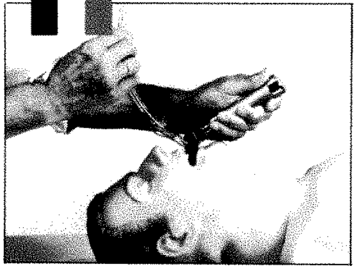

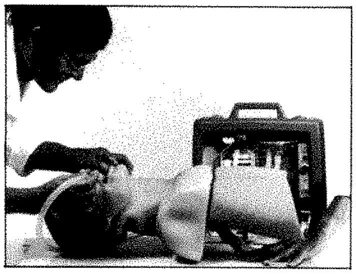

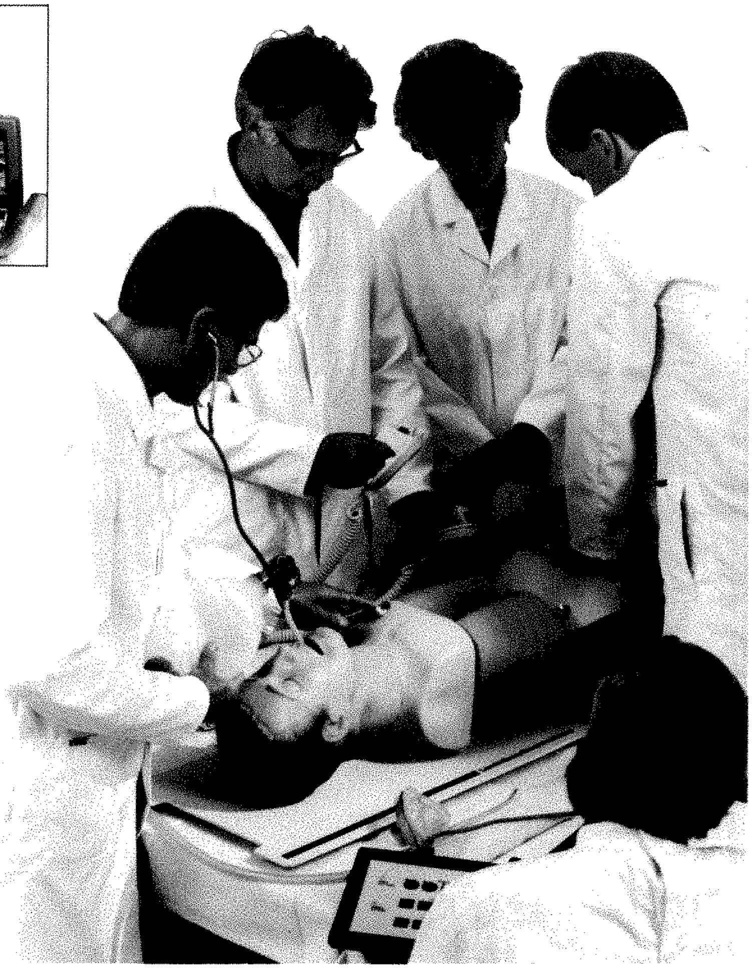

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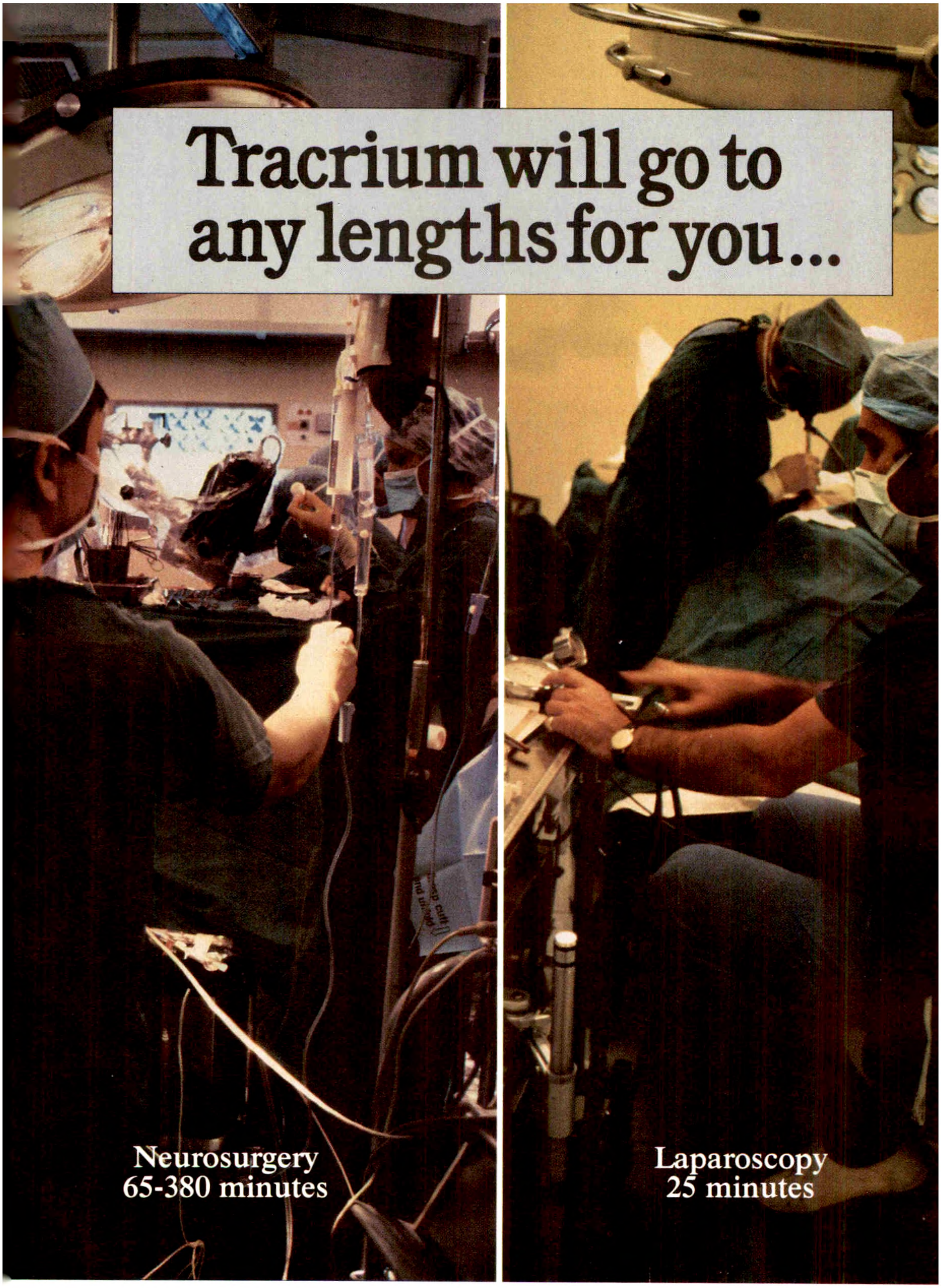
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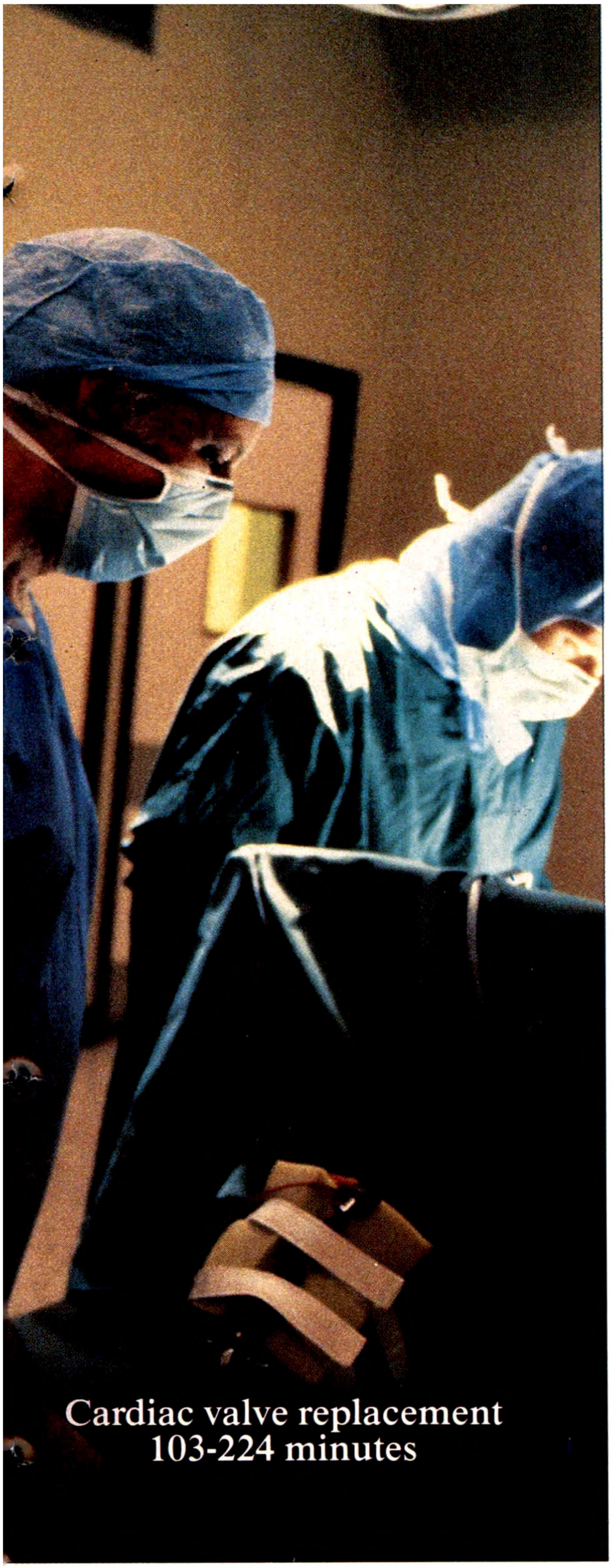

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Tracrium Prescribing Information

Uses Tracrium is a highly selective, competitive (non-depolarising) muscle relaxant for use in a wide range of surgical procedures and to facilitate control of respiration. Tracrium is highly suitable for endotracheal intubation, especially where subsequent relaxation is required.

Dosage and administration

Intravenous injection: Adults and children over 1 month 0.3-0.6 mg/kg depending on the duration of block required; this will provide relaxation for about 15-35 minutes. Tracrium may be used at standard dosage in elderly patients and in those with respiratory, hepatic or renal failure. Full block can be prolonged with supplementary doses of 0.1-0.2 mg/kg as required. Successive supplementary dosing does not give rise to cumulation. Endotracheal intubation can usually be accomplished within 90 seconds of intravenous injection of 0.5-0.6 mg/kg. The neuromuscular block produced by Tracrium can be rapidly and permanently reversed by standard doses of neostigmine which should be preceded by the administration of atropine. Recovery from full block without the use of neostigmine occurs in about 35 minutes.

Continuous infusion: Tracrium is suitable for administration by continuous infusion at rates of 0.005-0.01 mg/kg/minute (0.3-0.6 mg/kg/hour) to maintain neuromuscular block during long surgical procedures. Tracrium maintains acceptable physical and chemical stability in daylight at concentrations of between 0.5 and 0.9 mg/ml at 30°C for up to: 4 hours in: Compound Sodium Lactate Intravenous Infusion B.P.; 8 hours in: Ringer's Injection U.S.P.; Glucose Intravenous Infusion B.P. 5% w/v; Sodium Chloride 0.18% w/v and Glucose Intravenous Infusion B.P. 4% w/v and up to 24 hours in: Sodium Chloride Intravenous Infusion B.P. Tracrium can be administered by infusion during cardiopulmonary bypass surgery, at the recommended infusion rate. Induced hypothermia to a body temperature of 25 to 26°C reduces the rate of inactivation of atracurium, therefore full neuromuscular block may be maintained by approximately half the original infusion rate at these temperatures.

Contra-indications, warnings, etc. Contra-indicated in patients hypersensitive to atracurium besylate. Tracrium should be used with caution in patients receiving aminoglycosides or polypeptide antibiotics and in patients with myasthenia gravis. In common with all other neuromuscular blocking agents, adequate facilities must be available for endotracheal intubation and artificial ventilation. The neuromuscular block produced by Tracrium may be increased by the concomitant use of inhalational anaesthetics such as halothane. Tracrium should not be mixed in the same syringe with any other agent; in particular it must not be mixed with thiopentone or any alkaline agent, as the high pH would cause inactivation of the Tracrium. As with all other neuromuscular blocking agents the possibility of transient hypotension due to histamine release cannot be excluded. Although animal studies have indicated that Tracrium has no adverse effects on foetal development, nevertheless, like all neuromuscular blocking agents, it should be used with caution in pregnant women. Tracrium may be used to maintain neuromuscular relaxation in Caesarean section as atracurium does not cross the placenta in clinically significant amounts.

Presentation Tracrium injection contains 10 mg per ml atracurium besylate. Two ampoule sizes, 2.5 ml and 5 ml are available.

Basic NHS costs £6.50 for a box of 5 x 2.5 ml ampoules. £12.50 for a box of 5 x 5 ml ampoules. (PL3/0166).

Further information is available on request.

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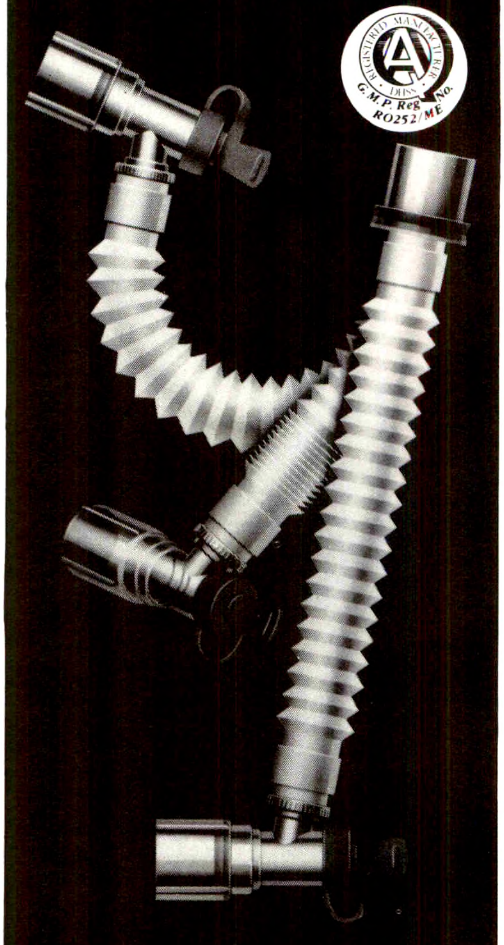

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Sir Ivan Whiteside Magill

[*Photograph by David Tredinnick, FRPS, AIMBI*]

Anaesthesia, 1987, Volume 42, pages 231–233

Editorial

Sir Ivan Whiteside Magill

KCVO, DSc (Hon), MB, BCh, BAO, FRCS (Hon), FFARCS (Hon), FFARCSI (Hon), DA

23 July 1888–25 November 1986

Ivan Whiteside Magill was born in Larne, Co. Antrim, on 23 July 1888, the son of Samuel Magill, a draper, second husband of Sara *née* Whiteside. It was, one presumes, a busy household since the birth was not registered until 7 September 1888. He was christened Ivan, not to honour a Russian ancestor, but because his mother thought that it sounded more romantic than John.

He was educated at Larne Grammar School, proceeded to Queen's University, Belfast, and qualified MB, BCh, BAO in 1913 with, and it gave him much quiet amusement in later years, a certificate to the effect that he had given one anaesthetic. He was to give his last in 1972, having contributed more to the practice of anaesthesia and its establishment as a specialty than anyone.

After qualification he became House Surgeon and then Resident Medical Officer at the Stanley Hospital, Liverpool, and was RMO at the Walton Hospital when the Great War broke out in 1914. He left that post after holding it for 6 months, took a temporary commission in the Royal Army Medical Corps, and served throughout the war with the rank of Captain: he was Medical Officer to the Irish Guards at the Battle of Loos.

In 1916 he married Edith Banbridge who was also a doctor. They were to have no children. She died in 1973.

At the end of hostilities he was posted to Barnet War Hospital where, among other duties, he gave occasional anaesthetics for two surgeons. To a questionnaire from the RAMC asking for his current status with demobilisation in view, he answered 'Anaesthetist' (put up to it, as he said later, by his colleagues) and early in 1919 he was posted to Queen Mary's Hospital for Facial and Jaw Injuries at Sidcup. On arrival he met Stanley Rowbottam, coincidentally posted. Thus began an historic relationship with two inexperienced young men facing frightful anaesthetic problems for the handling of which there was little precedent.

Harold Gillies and Pomfret Kilner were the surgeons and most of the surgery was for the repair of gross facial trauma, the residuum of 4 years of warfare. Anaesthesia was with rectal ether, oral airways and Silk's tubes, or intratracheal air and ether insufflated through a catheter. These procedures were hazardous and insecure, surgeon and anaesthetist in each other's way, and ether-laden air inhaled by the surgeon after it had bubbled through the operative field.

Magill devised his forceps for the introduction of nasal catheters into the larynx under direct laryngoscopic vision but sometimes they entered the larynx without instrumentation and, intrigued by this, he practised 'blind' introduction.

The fruit of his interest and practice culminated in a particular anaesthetic which he was to remember for the rest of his life. A patient was to have an anaesthetic which he decided could only be continuous nitrous oxide. He passed a wide-bore rubber tube through the nose and into the larynx through which she breathed to and fro, inhaling and exhaling from a constant flow apparatus with a reservoir bag and exhaust valve. In one step, Magill had secured a safe and adequate airway, removed himself from the operative field and made possible the use of nitrous oxide for a long operation. To use it for insufflation had been much too extravagant. He said 'I confess it came to me as a revelation'.

He later was to describe the ideal position of the patient's head for this manoeuvre to be as of a man 'scenting the air'. It was, however, a subsequent simile which was more characteristic—'draining a pint of beer'.

In 1923 he was appointed to the Brompton Hospital. Chest disease was then gross and tuberculosis, for which there was no specific therapy, was rife; he applied himself with the flair which he had

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already shown at Sidcup, to anaesthetise patients with copious sputum. He devised a suction with an inflatable cuff as a bronchial blocker, so that secretions could be aspirated from t being operated on, and endobronchial tubes with bronchoscopic introducers for one-lung anae Chest surgery became ever more ambitious as his efforts made this possible.

In 1924 he joined the Westminster Hospital where he worked with, among other surgeons, S Cade who was later to write '... to have Magill as one's anaesthetist put the surgeon in a " That class included Harold Gillies, Tudor Edwards and Terence Millin.

From the early days at Sidcup onwards it was inevitable that he would be greatly in den private practice. His ingenuity found many ways to ensure that he could give a safe anaesthe apparatus that he could carry and use in a private house, in one of the ill-equipped nursing h the day or even, 'for 4 hours in a bathroom in the Ritz'.

He made in 1920 a portable drip-feed ether apparatus with a foot bellows. He placed batt the handle of his laryngoscope. In 1926 he added nitrous oxide and a dry gas flow mete apparatus (his cylinders he carried under his overcoat suspended from a yoke over his shoulde intermittent flow machine for dental gases followed and, much later, he was quick to see the ance of the Rotameter. He saw problems and needs clearly, and he found simple answers.

In 1930 he returned from the United States with Nembutal and in 1952 with Arfonad. H papers on his own apparatus and techniques and wrote chapters on anaesthesia for textbooks by his surgical colleagues.

It would have been false humility for him not to have realised the immense contribution made to the practice of anaesthesia but, towards the end of his life, he was to say that he w proud of the part he had played in the establishment of anaesthesia as a specialty. He wrote, ' took up anaesthetics, I felt that the importance of the service would never be realised until it recognised as a specialty with a Diploma entailing the necessary training and study.'

In 1931, as Senior Secretary of the Anaesthetic Section of the Royal Society of Medic proposed to the Council that an examination for a Diploma in anaesthesia should be ins Council was told by the Secretary of the Society that the RSM, an academic body, could no its charter, initiate this. It was decided, after considerable deliberation, to found an associa anaesthetists and this was constituted on 1 July 1932 to concern itself with the political rath the scientific aspect of anaesthesia. The first examination for the Diploma of Anaesthetics Conjoint Board of the Royal College of Surgeons of England was held on 8 November 1935. the world's first qualification in anaesthesia. Throughout the negotiations Magill was as dete resourceful and tireless as he was in the practice of anaesthesia; with John Challis and Joe Bl he decided what experience a would-be candidate should have before entering for the exam and he was on the first board of examiners. His standards were, however, too high: woe bet unhappy candidate who, recognising him, suggested unnecessary intubation. His fellow exa soon persuaded him that he had 'done his bit'.

The respect in which he was increasingly held, was made manifest by many honours.

In 1937 he was the President of the Section of Anaesthetics of the Royal Society of Medi 1938 he was awarded the Henry Hill Hickman medal and in 1939 the medal of the Ulster Society.

He worked in London throughout the Second World War, was consultant adviser to the Eme Medical Service and Civilian Consultant to the Royal Navy.

In 1945 Belfast University made him a Doctor of Science (having earlier rejected his M.D based on his most famous achievement—blind nasal intubation).

In 1946, for service in high places, he was made a Commander of the Royal Victorian Order 1960 he was knighted.

He was made an Honorary Fellow of the Faculty of Anaesthetists of the Royal College of Su which was founded in 1948, of the Royal Society of Medicine and of the Royal College of Su

In 1963 he was made a member of the British Association of Plastic Surgeons; anaesthetis plastic surgeons in the United States honoured him likewise. In 1966 he expressed his thanks recipient of the Ralph Waters Award and he quoted Waters, 'In our struggle to become scien must guard against the real danger of losing our art.' He was in no danger of doing that.

Until his retirement from the Health Service he continued to work at Westminster Hospital where, as 'the Boss', 'the old man' or simply 'Maggi' (his preferred spelling) to generations of junior anaesthetists (and some senior ones), his Monday and Tuesday afternoon sessions were a Mecca. His Rabelaisian asides, delivered in what he thought was a whisper, but audible for several feet around, were much relished. He never taught save by example and it was obvious that, no matter how effortless or even casual his work might seem, every anaesthetic was the best he could give, having made sure by meticulous preparation, that he would never be let down by his apparatus. Magill was a superb anaesthetist with an extraordinary deftness and an air of unhurried purpose. Nothing, one felt, could surprise him and nothing could go wrong. His hand never left the bag and years of the keenest observation had endowed him with an almost uncanny feel for the condition of a patient. His memorial at Westminster Hospital Medical School is the Magill Department of Anaesthetics.

Kilner remembered him as a student 'of enviable physique, broad in the shoulders, unusually strong and muscular in the arms, keen on boxing (practised on the local police) and tennis'. He had, in fact, boxed for his university as a heavyweight and was a rugby forward.

He cherished his toughness throughout his life and was proud of a fitness which he carefully maintained. He was an eminently clubbable man, a lover of food, drink, lively company and a joke—especially a practical one—and his presence was forever betrayed by the aroma of the Burma cheroots which were almost his trade mark.

With juniors he was generous and effortlessly unaffected, very much a man's man and, without any condescension, 'one of the boys'. Kindly, straightforward and immensely loyal to friends and to his specialty, he retained the simple morality of his youth; one must never 'let the side down'.

His passion and joy was fishing at which, as in most things, he excelled. In 1932 he had been made a member of the most exclusive of clubs, The Houghton, at Stockbridge on the Test and here almost every spare moment was spent relentlessly stalking trout, while his wife sketched. He caught a five-pounder on his 97th birthday.

Reminiscing about bygone friends, particularly fishermen, his greatest accolade, frequently bestowed, was 'he was a hulluva decent fella'. Of no one could this more truly be said than of himself.

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Editorial notices

Submission of manuscripts

Manuscripts must be submitted in accordance with the internationally recognized *Uniform requirements for manuscripts submitted to biomedical journals* (*British Medical Journal* 1979; 1: 532–5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

Anaesthesia News

A new service for members of the Association of Anaesthetists of Gt Britain and Ireland is announced. This news sheet will be distributed free with members' copies each quarter. It replaces the News and Notices section of the Journal, with the exception that Obituarial Notices and the International Calendar will continue to be published quarterly. Publication of the Annual Report of Council and Linkman Conferences will also continue.

Instructions for submissions to *Anaesthesia News* are included in the first number, to appear with the April issue of *Anaesthesia.*

Anaesthesia, 1987, Volume 42, pages 234–242

Fluorocarbons

Current status and future applications

N. S. FAITHFULL

Summary

This article presents information on medical applications of fluorocarbons. These inert chemicals have a high solubility for the respiratory gases and, in emulsified form, are present in the oxygen-transporting plasma substitutes now undergoing clinical trials. Oxygen content is directly related to arterial partial pressure of oxygen ($Pa\text{O}_2$). Thus, although oxygen transport of Fluosol-DA 20% (FDA) is only 0.75 ml per 100 ml per 10 kPa, oxygen delivery at the tissues will be adequate in the presence of a high $Pa\text{O}_2$. FDA has a low viscosity at low shear rates and this, together with a very small emulsion particle size of 0.1 μm, helps provide improved tissue oxygenation in conditions of ischaemic hypoxia. Fluorocarbon plasma substitutes may be employed as 'blood', but may also be used for a wide range of purposes in clinical practice. This review describes some current and potential future applications.

Key words

Pharmacology; fluorocarbons.

The development of blood substitutes capable of carrying and delivering substantial amounts of oxygen, has proceeded along two different lines. Stroma-free haemoglobin solutions suffer from a short half-life in the circulation (approximately 20 minutes) and a P_{50} lower than normal blood. The latter will decrease oxygen availability in the tissues, while the high osmotic pressure of haemoglobin solutions limits both the concentration that can be used and the total oxygen flux. These problems are being overcome in various ways, such as micro-encapsulation with synthetic cell membranes[1] or polymerisation of the haemoglobin molecule.[2]

The other approach to the production of oxygen-transporting plasma substitutes involves the use of fluorocarbons. These substances are often known as perfluorocarbons or, more correctly, perfluorochemicals (PFCs); this paper briefly describes their properties and possible clinical applications.

Fluorocarbon-containing blood substitutes

The physiological availability of oxygen dissolved in PFCs was dramatically demonstrated by Clark and Gollan,[3] who were able to show survival of mice completely immersed in these liquids for extended periods of time.

Fluorocarbons can dissolve more than 50 volumes percent of oxygen and more than 400 volumes of carbon dioxide,[4] but are immiscible with blood. To avoid embolic phenomena emulsions are used, and the Green Cross Cor-

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Table 1. Composition of Fluosol-DA 20% and FC-43. Constituents are given in weight per volume percent.

	Fluosol-DA 20%	FC-43
Perfluorotributylamine	—	20
Perfluorodecalin	14	—
Perfluorotripropylamine	6	—
Pluronic F68	2.7	2.56
Yolk phospholipids	0.4	—
Glycerol	0.8	—
NaCl	0.6	0.6
KCl	0.34	0.34
$CaCl_2$	0.028	0.028
$MgCl_2$	0.02	0.02
$NaHCO_3$	0.21	0.21
Glucose	0.18	0.18
Hydroxyethyl starch	3.0	3.0

poration of Osaka, Japan has for some years produced such a blood substitute, containing 20% fluorocarbons by weight (13.4% by volume). This product is known as Fluosol-DA 20% (FDA). Another product produced by the same firm is known as FC-43, and contains perfluorotributylamine, the PFC originally used in the classical experiments of Geyer *et al.*[5] who demonstrated survival of rats in which practically all blood had been replaced by PFC emulsion.

The compositions of FDA and FC-43 are shown in Table 1. The Pluronic F68 and egg yolk phosphatides are present as emulsifying agents and the resulting very fine emulsion has a mean particle size of approximately 0.1 μm.[6] Hydroxyethyl starch (HES) provides the osmotic pressure of the preparation. The glycerol present as a stabiliser is almost immediately metabolized.[7] FDA has poor room-temperature emulsion stability and is supplied frozen. Immediately prior to use it is thawed out and two annex solutions must be added.

A number of other fluorocarbons are currently undergoing trials as candidates for 'second generation' products in Japan, the United States and France. FDA, which is undergoing clinical trials, may not enter widespread clinical practice but the arrival of some fluorocarbon-containing preparation is not far distant.

Oxygen transport by fluorocarbon-containing preparations

The oxygen carried by PFCs is directly proportional to the oxygen tension (Fig. 1). Owing to the S-shaped oxyhaemoglobin dissociation curve, this is not so in the case of haemoglobin and uptake and release of oxygen take place over a very narrow physiological range.

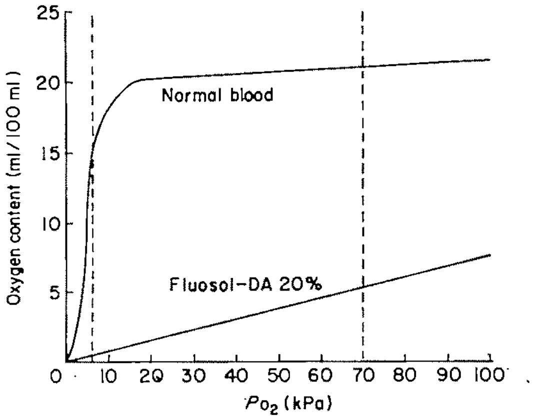

Fig. 1. The relationship between oxygen partial pressure in normal human blood and Fluosol-DA 20%.

At full saturation, 100 ml of normal blood contains approximately 20 ml of oxygen at a $P\mathrm{o}_2$ of about 16 kPa. At this tension, FDA will contain approximately 1.2 ml of oxygen per 100 ml of emulsion. This amount, though small, is nevertheless more than three times as much as can be carried by plasma alone. At a partial pressure of oxygen of 70 kPa, a tension reasonably obtainable while 100% oxygen is breathed in the absence of pulmonary disease, FDA still contains only about 5.25 ml of oxygen per 100 ml.

In Fig. 1, the vertical dotted line at a $P\mathrm{o}_2$ of 6 kPa represents tissue $P\mathrm{o}_2$. It is, of course, almost impossible to quantify tissue $P\mathrm{o}_2$, which is influenced by the supply/demand situation in each organ and may vary widely between closely adjacent areas in the same tissue.[8,9] When the $P\mathrm{o}_2$ is decreased from 70 to 6 kPa, 4.8 ml of oxygen are released from FDA. This is only slightly less than the 5.47 ml released by normal blood over the same range.

In practice, in an individual whose circulation contains both blood and Fluosol, the high oxygen extraction from fluorocarbons will result in a far greater oxygen delivery than might have been expected from oxygen transport dynamics. In an early clinical case report, for instance, fluorocarbons accounted for 27% of total oxygen flux but provided more than 50% of the oxygen consumed.[10]

Elimination and toxicity

The half-life of Fluosol-DA is very species- and dose-dependent.[11] Yokoyama *et al.*[12] calculated a half-life of 7.5 hours after administration of 10 ml/kg body weight and of 14.5 hours following 20 ml/kg. The value rose to 22 hours after 30 ml/kg.

PFCs are excreted primarily via the lungs.[13] They are not metabolised *in vivo*, owing to the large value of the carbon–fluorine bond strength of 116 kcal/mol.[14] This was confirmed by the lack of increase in plasma fluoride following administration in man.[15]

Fluorocarbons are taken up in significant amounts by the reticulo-endothelial system (RES). Of those present in FDA, perfluorodecalin (FDC) is taken up much less than perfluorotripropylamine (FTPA). The latter improves the stability of the FDC, which would otherwise form larger emulsion particles which result in increased toxicity.[16] Okamoto *et al.*[17] showed that FDC was almost completely excreted from mice after 6 weeks, whereas FTPA was still to be found in significant quantities in the liver and spleen. Studies on human autopsy material indicate complete removal of PFCs within 7 months.[18] Animal studies have also revealed changes in serum enzyme concentrations following very high rates of administration of FDA, and reticular cell proliferation in liver and spleen was accompanied by coarse intracellular vacuolation.[19]

Elevations in SGOT and SGPT enzyme levels in four out of eight patients who received Fluosol were attributed to antibiotics received during surgery.[20] In a large Japanese trial,[21,22] increases in SGOT were seen in only eight patients out of a total of 122. These changes may imply hepatic toxicity, but they may merely indicate intrahepatic obstruction caused by rapid uptake of PFCs by Kupffer cells.

Accumulation in the RES undoubtedly occurs, but very little evidence has been presented that this is actually harmful in the long term. Rosenblum *et al.*[23] performed autopsies on Macaque monkeys and observed accumulation of PFCs and vacuolation of Kupffer cells in the RES that persisted for 6 years after administration of FC-43 emulsions. These room-temperature stable emulsions are never used clinically and are known to accumulate extensively in the RES. No PFCs were observed in monkeys that had received perfluorodecalin 8 years previously. Following both agents, there appeared to have been a long-term stimulation of the RES that resulted in increased production of immunoglobulins.

Short-term increase in pulmonary pressure, decreased leucocyte count and occasionally chest pain have been described when susceptible patients were challenged with Fluosol,[24] and studies have shown that both Fluosol and the emulsifier Pluronic F-68 can activate plasma complement.[25] In rabbits[25] and humans[26] this reaction can be blocked by pretreatment with methylprednisolone. The above reactions have almost all been reported in Caucasian patients and no significant increases in leucocyte counts and decreases in complement were observed when Japanese patients were challenged with Fluosol.[27] It has been suggested that this may be due to the differing diet of the two peoples.

Clinical studies

FDA was first administered clinically to five decerebrate (brain dead) subjects.[28] Twenty-four hours later, autopsy revealed vacuolation of Kupffer cells in liver and spleen, although no changes in serum transaminases had been noted. Following this, Fluosol was administered to ten healthy volunteers.[11] No adverse reactions were noted. Administration of Fluosol-DA 20% to eight surgical patients[20] revealed slight increases in circulating blood volume, with increased antidiuretic hormone levels. No significant changes were seen in aldosterone, cortisol, insulin or growth hormone levels. Increases in pulmonary artery pressure, capillary wedge pressure and cardiac output following Fluosol were significantly more than following whole blood, 6% HES or 3% dextran 40.[29] The $P\mathrm{ao}_2$ increased and calculated shunt fractions decreased. Fujita *et al.*[30] have shown that postoperative recovery of RES function may be slightly delayed by Fluosol as compared with HES, as assessed by the phagocytic index. However, no increase in immune suppression was observed.

The oxygen delivery efficacy by FDA may be indicated by increased oxygen consumption, decreased cardiac index or increased mixed venous oxygen content.[31] Its consumption and its contribution to oxygen flux can be calculated from $P\mathrm{ao}_2$, $P\mathrm{vo}_2$, the fluorocrit (percentage of fluorocarbons in the blood determined by high-speed centrifugation) and cardiac output.

In a Japanese trial, Fluosol was administered to 186 patients.[22] Unfortunately, no details of cardiac output were given, but it was calculated that fluorocarbon-transported oxygen accounted for 11.7% of the total body oxygen consumption at a mean fluorocrit of 3.3% and a mean Pao_2 of 26.3 kPa (a value at which Fluosol would not carry much oxygen). Tremper *et al.*[32] reported that 20 ml/kg FDA accounted for 7% of the oxygen flux and 24% of the oxygen consumption at mean fluorocrits of 2.9% and Pao_2 values of 48.0 kPa. Similar phenomena have been noted by Watanabe.[33] Others[34] have concluded that Fluosol might allow reduction of FIO_2 if the patient required 100% O_2 to maintain oxygen transport at acceptable levels. Clinical trials in the use of Fluosol in grossly anaemic patients (Jehovah's Witnesses who have refused blood on religious grounds) are proceeding in the USA, although the American Food and Drugs Administration (FDA) has not yet permitted release of the drug.[35]

Microcirculatory support by fluorocarbon emulsions

One of the most potentially beneficial therapeutic uses for FDA may be as a supporter of the ischaemically hypoxic microcirculation. PFC emulsions have a very low viscosity which, unlike that of blood, hardly alters as shear rate decreases in the microcirculation (Fig. 2). *In vivo* studies[36] have confirmed that PFC emulsions have a much lower viscosity than blood. This implies a higher tissue blood flow for the same blood pressure, and it has been shown[37] that the oxygen carrying capacity of FDA is sufficient to allow replacement of 30% of blood volume with the emulsion and still maintain normal whole body oxygen flux; this was true even in the presence of a moderate sized fresh myocardial infarct.

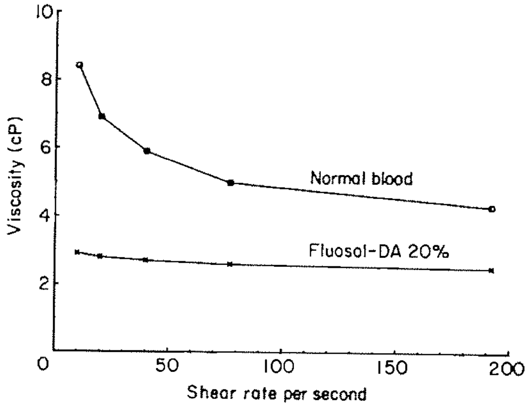

Fig. 2. The viscosity of normal human blood and Fluosol-DA 20%.

Biro[38] has suggested that, in conditions such as myocardial infarction, reduction in viscosity by haemodilution may improve perfusion through available collaterals. Others[39] have maintained that the small PFC particles may bypass obstructions in the circulation caused by endothelial blebs and sludged red cells. Still others[40,41] have maintained that the particles can penetrate deep into hypoxic tissue beds, not only bypassing sludged cells but re-oxygenating them and reversing the stiffening of the red cell membranes which occurs under conditions of hypoxia and tissue acidosis.[42,43] Hence, the vicious circle of sludged standstill can be broken.

PFCs may also affect the outcome of ischaemic hypoxia by their actions on leucocytes, because they can inhibit neutrophil phagocytosis, chemotaxis and superoxide release.[44,45] These help explain the decreased inflammatory response and increased myocardial salvage observed following early postinfarction haemodilution with PFCs.[46] This mechanism would also explain the ability of PFCs to protect against the reperfusion syndrome.[47,48]

Assessment of myocardial infarct size following vascular occlusion, indicates that treatment with PFC emulsions improves flow to the ischaemic area[38,49] and often increases local oxygen supply.[49,50] These findings have been confirmed by studies which show that local intramyocardial oxygen tensions may be increased in the infarcted area by treatment with PFCs in both dogs[50] and pigs.[51] These changes result in reduction in infarct size,[38,49] particularly in relation to the volume of myocardium at risk.[52,53,46]

Reports have recently appeared[54,55] in which PFCs have been used successfully during percutaneous transluminal coronary angioplasty. Oxygenated FDA was perfused through the central lumen of the catheter to provide oxygenation to the distal myocardium and this successfully reduced ST segment changes and allowed prolongation of ballon occlusion times.

In a middle cerebral artery occlusion model, it was found that Fluosol treatment could lessen cerebral damage,[56] although it was impossible to provide complete protection.[57] Administration of the oxygen radical scavenger mannitol following PFC administration,[58] or treatment with both agents prior to ischaemia,[59] improved results. Subarachnoid space perfusion with oxygenated fluorocarbon nutrient solution can cause marked improvement following severe cerebral ischaemia[60] or spinal cord injury.[61]

A report on the use of Fluosol in 73 patients who suffered from cerebral ischaemia of varying aetiology,[62] has shown clinical improvement in about 65% of cases; in 20 of the patients, cerebral blood flow was increased by a mean of 20%. In another clinical report,[63] patients were treated with Fluosol and mannitol before undergoing cerebral revascularisation procedures. Neither study contained control groups and, alas, no firm conclusions can be drawn from these studies.

The use of PFC emulsions for the treatment of sickle cell crisis is feasible and *in vitro* experiments[64] have confirmed oxygen transfer to sickled erythrocytes and subsequent reduction of blood viscosity, particularly at low shear rates. The high penetrability and small particle size of PFCs should help 'loosen up' the circulation *in vivo*.

The use of fluorocarbon emulsions may also be of benefit in conditions of incipient gangrene in peripheral vascular disease.[65] Whether the mechanism whereby intraluminal PFCs can improve intestinal ischaemia[66] is the result of particle absorption into the circulation or of oxygen diffusion across the gut is not clear, although it is possible to supply oxygenation by the latter mechanism.

Another potentially important application of PFCs is their ability to improve circulation and hence oxygenation in the centre of solid tumours. Recent work has confirmed that treatment with PFCs and oxygen before[67,68] and during irradiation[68] can improve radiosensitivity of hypoxic tumours. Improvement of tumour circulation may also facilitate the penetration and efficacy of cytotoxic agents.

Recent work[69] has suggested that haemodilution with FDA may reduce critical oxygen delivery levels during haemorrhagic shock and thus protect against tissue hypoxia under these conditions. This was possible even in the absence of oxygen enrichment of the inspired air and may be due to the ability of PFCs to promote oxygen diffusion across the endothelial membrane.

Other applications of fluorocarbons

Perfusion with oxygenated PFC emulsions for preservation of transplantation organs has been performed frequently and the first reports involved brain perfusion.[70] Good maintenance of function has been reported for PFC-perfused kidneys in experimental animals[71] and humans[72,73] with subsequent successful re-implantation. Hearts have been perfused for up to 2 hours at 37°C and successfully transplanted,[74] and successful short-term perfusion of the liver has been carried out with good maintenance of protein synthesis.[75] Traumatically amputated human extremities have been successfully perfused for up to 72 hours and successful re-implantation has been achieved.[41]

Perfluorochemicals are radio-opaque[76] and are under investigation as contrast agents in radiography. They have been used in bronchography in both animals and humans[77] and, in combination with computer tomography, have been used for experimental enhancement of hepatic, splenic and vascular structures.[79] Prior PFC administration may also help in the ultrasonic localisation of hepatic tumours.[79] Oxygenated FDA has been employed in clinical coronary angiography and has been found to lead to less electrocardiographically detected myocardial ischaemia than when conventional agents were used.[80]

Reports of the successful use of PFC-containing liquid membrane oxygenators during partial bypass have appeared[81] and total cardiopulmonary bypass with PFCs was feasible in dogs,[82] although this rapidly led to uptake of fluorocarbon particles in the liver.[83] Studies that used PFC emulsions as cardioplegic solutions during prolonged global ischaemia,[84] indicated superiority over administration of crystalloid or oxygenated blood, as judged by increases in myocardial oxygen tension and oxygen consumption.

PFC emulsions may be used outside the blood stream to improve whole body gas exchange, for instance during respiratory failure. Arterial oxygen tensions were raised and venous carbon dioxide tensions lowered by perfusion of the peritoneal cavity of rabbits[85] and rats[86] with oxygenated FDA. More than 15% of oxygen consumption can be provided by this route.[87]

Fluorochemicals are not altered in the body, but they do appear to have some biological properties. Single intraperitoneal or intravenous administration of PFCs can augment the tumouricidal potential of peritoneal macrophages for as along as 14 days[88] and may induce antimicrobial activity and increased survival of rats with induced *E. coli* peritonitis.[65] Similarly, transtracheal fluorocarbons improve survival in experimental *Pseudomonas* pneumonia.[65] The above properties may be very useful in a clinical situation.

There are a number of other interesting and potentially useful clinical applications for fluorocarbons which are too numerous to discuss in this article. Suffice it to say that the future use of these compounds will not be limited to their employment as 'artificial blood', a term so beloved of the media, but in a wide and divergent spectrum of multidisciplinary uses. It is hoped that this article has given some insight into these applications.

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Anaesthesia, 1987, Volume 42, pages 243–248

Acute beta-adrenoreceptor blockade and induced hypotension

D. L. SIMPSON, W. R. MACRAE, J. A. W. WILDSMITH AND B. A. B. DALE

Summary

Thirty patients scheduled for major middle ear surgery and induced hypotension, with a trimetaphan/sodium nitroprusside infusion, were randomly allocated to receive a single oral dose of beta-adrenoreceptor antagonist 2 hours before surgery. In group 1 ten patients received metoprolol 50 mg, in group 2 ten patients received metoprolol 25 mg and in group 3 ten patients received oxprenolol 20 mg. There were ten controls for each group. The nitroprusside infusion rate was significantly reduced in groups 1 and 3. However, there was an unacceptably high incidence of profound bradycardia after induction of anaesthesia in those patients who had received beta-adrenoreceptor antagonists pre-operatively and this technique is not recommended for routine use.

Key words

Anaesthetic techniques; induced hypotension.
Sympathetic nervous system; beta-adrenergic antagonists.

Induced hypotension is used in many centres as part of a general anaesthetic technique intended to improve surgical access during procedures such as middle ear surgery. Various agents are employed to reduce blood pressure and the failure of any one to gain universal acceptance indicates the difficulties in producing consistent results. In our hospital hypotension is induced during halothane anaesthesia by an infusion of a mixture of trimetaphan camsylate and sodium nitroprusside. This technique, which combines the properties of sodium nitroprusside and trimetaphan in a synergistic fashion, provides a readily controllable degree of hypotension while cardiac output is maintained in the large majority of patients.[1,2]

Review of the records of 200 patients who received this combination, showed that hypotension was readily induced and controlled in 187 cases (93.5%). In 40 patients (20%) there was no change in heart rate and in 147 (73.5%) there was a moderate increase which was not associated with any difficulty in blood pressure control. In the remaining 13 patients (6.5%) a combination of tachycardia and difficulty in the production of hypotension required the additional use of agents such as phentolamine and propranolol in an attempt to control blood pressure and provide good operating conditions. This resistance to induced hypotension was first noted many years ago, at the time of the introduction of the ganglion blockers into anaesthetic practice.[3] Progressive improvements in technique have decreased the incidence of the

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problem, but such patients continue to comprise a small, unpredictable but persistent group who cause considerable practical difficulty.

Khambatta *et al.*[4] have shown that treatment with propanolol (180 mg in three divided doses) in the 24 hours prior to surgery reduced the incidence of tachycardia and rebound hypertension after hypotension induced by sodium nitroprusside. The present study was designed to examine the effect of a single pre-operative dose of beta-adrenoreceptor antagonist on hypotension induced with a trimetaphan/sodium nitroprusside mixture.

Methods

Patients and anaesthesia

Sixty patients (aged 18–65 years, ASA grade 1 or 2) scheduled for major middle ear surgery were studied. The study was approved by the local ethical committee and the patients gave informed consent. The patients were premedicated with diazepam 10 mg orally. Anaesthesia was induced intravenously with tubocurarine 5 mg, thiopentone 350–500 mg and suxamethonium 60 mg. After tracheal intubation, anaesthesia was maintained with nitrous oxide 40%, oxygen 60% and halothane 1.5–2%. The patients breathed spontaneously after the return of neuromuscular function. Both the electrocardiogram and arterial pressure (measured directly with a radial artery cannula) were monitored continuously.

Once anaesthesia was stable, the patient was placed in a 5° head-up position, and an infusion of trimetaphan 50 mg and sodium nitroprusside 12.5 mg in 500 ml of 5% dextrose was commenced, with the aim of producing a systolic arterial pressure between 55 and 60 mmHg.

Study protocol

There were three groups of 20 patients: group 1 comprised ten controls and ten subjects who received metoprolol 50 mg 2 hours pre-operatively (orally); group 2 comprised ten controls and ten subjects who received metoprolol 25 mg 2 hours pre-operatively (orally); group 3 comprised ten controls and ten subjects who received oxprenolol 20 mg 2 hours pre-operatively (orally). Heart rate and blood pressure were recorded prior to anaesthetic induction, 5 minutes after induction and 20 minutes after starting the hypotensive infusion. The total volume of the mixture given was recorded to allow calculation of the sodium nitroprusside infusion rate for each patient (μg/kg/minute).

The operative field was assessed by one surgeon, who was unaware of the technique used and scored the conditions on a visual analogue scale between limits of 10 (ideal) and 0 (inoperable).

Results

The results are shown in Figs. 1–5. Statistical

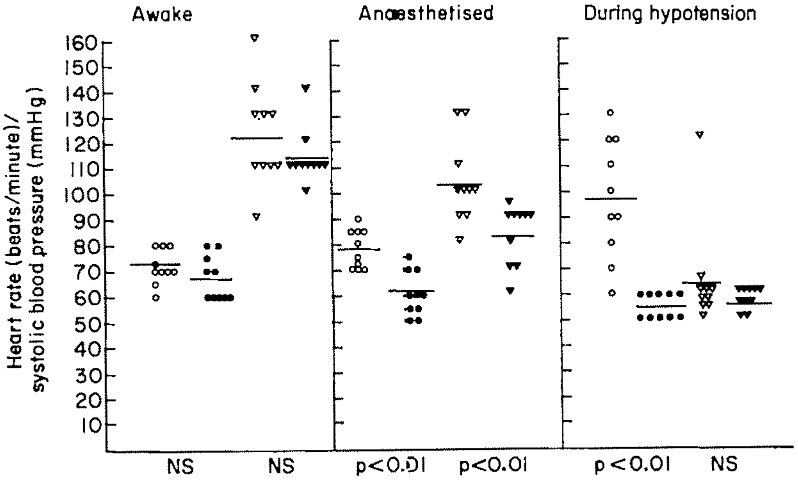

Fig. 1. Heart rate and systolic blood pressure in group 1 (metoprolol 50 mg). *Control group:* ○, heart rate; ▽, systolic blood pressure. *Pre-operative beta blockade:* ●, heart rate; ▼, systolic blood pressure. Horizontal bars indicate the mean values.

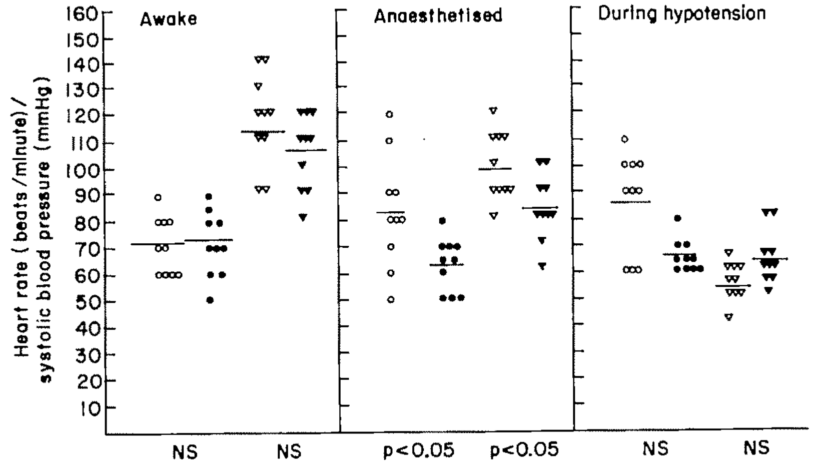

Fig. 2. Heart rate and systolic blood pressure in group 2 (metoprolol 25 mg). *Control group:* ○, heart rate; ▽, systolic blood pressure. *Pre-operative beta blockade:* ●, heart rate; ▼, systolic blood pressure. Horizontal bars indicate the mean values.

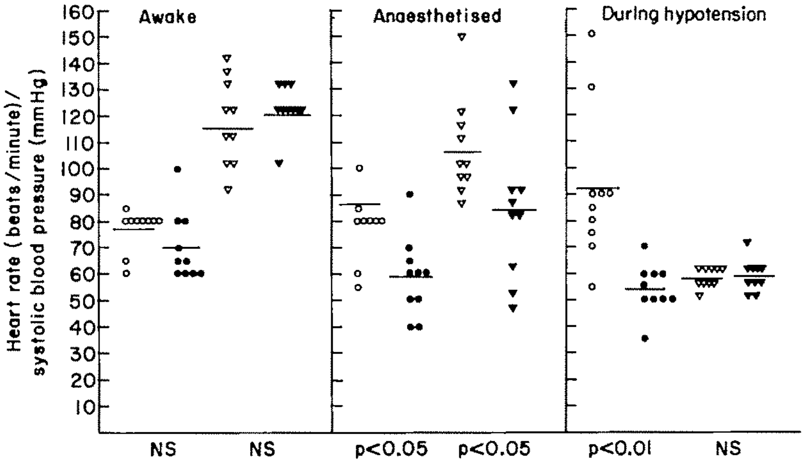

Fig. 3. Heart rate and systolic blood pressure in group 3 (oxprenolol 20 mg). *Control group:* ○, heart rate; ▽, systolic blood pressure. *Pre-operative beta blockade:* ●, heart rate; ▼, systolic blood pressure. Horizontal bars indicate the mean values.

comparisons were made using the Wilcoxon rank sum test.

Haemodynamic data (Figs. 1–3)

After the induction of anaesthesia, those patients who had received beta-adrenoreceptor antagonists pre-operatively had a significantly lower mean heart rate and systolic blood pressure than the controls. After 20 minutes of induced hypotension, those patients who had received metoprolol 50 mg or oxprenolol 20 mg had significantly lower heart rates than the controls.

Sodium nitroprusside infusion rate (Fig. 4)

There was a highly significant difference in the rate of infusion in those patients who had received metoprolol 50 mg or oxprenolol 20 mg, which indicates the greater ease with which the target blood pressure was obtained in those patients.

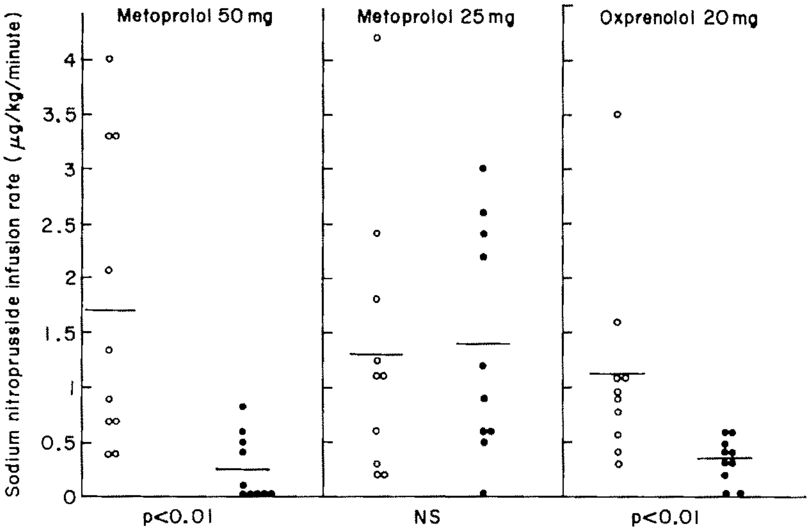

Fig. 4. Sodium nitroprusside infusion rate (μg/kg/minute). ○, Control group; ●, pre-operative beta blockade. Horizontal bars indicate the mean values.

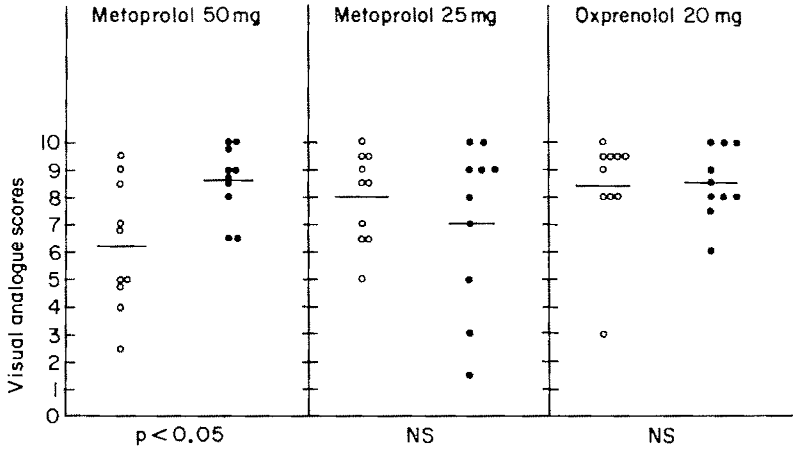

Fig. 5. Surgeon's visual analogue score of operating conditions (10 = ideal, 0 = inoperable). ○, Control group; ●, pre-operative beta blockade. Horizontal bars indicate the mean values.

Operating conditions (Fig. 5)

Only metoprolol 50 mg resulted in a significant improvement in operating conditions. It is important to note that the control patients in this group scored badly with regard to operating conditions and required the highest rate of trimetaphan/nitroprusside administration. No significant difference is found if the metoprolol 50 mg patients are compared with the control data from the other two groups.

Untoward effects

A number of anaesthetic problems were noted in the patients who received beta-adrenoreceptor antagonists (Table 1). The commonest was profound bradycardia after the induction of anaesthesia. Five patients required intravenous atropine because their heart rates decreased to less than 45 beats/minute, and in one patient in group 1 the profound bradycardia discouraged the induction of hypotension.

Table 1. Adverse effects in patients who received beta-adrenoreceptor antagonists.

Group	Controls	Pre-operative beta blockade
Group 1 (metoprolol 50 mg)		Bradycardia following induction (35/minute)—treated with atropine
Group 2 (metoprolol 25 mg)	Bradycardia following infusion of trimetaphan/SNP—treated with atropine	Bradycardia following induction—treated with atropine
Group 3 (oxprenolol 20 mg)	Supraventricular tachycardia following induction—resolved spontaneously Persistent sinus tachycardia (140/minute)—treated with intravenous propranolol	Bradycardia following induction—treated with atropine Bradycardia following induction—treated with atropine Bradycardia following induction aggravated by infusion of trimetaphan/SNP—treated with atropine

SNP, Sodium nitroprusside.

One patient in a control group had a bradycardia and another developed a supraventricular tachycardia after induction. This resolved spontaneously. A third control patient developed a sinus tachycardia of 140 beats/minute following the induction of hypotension. This required treatment with intravenous propranolol in 1 mg increments to a total of 3 mg.

Discussion

A decrease in blood pressure evokes reflex homeostatic mechanisms. Increased baroreceptor activity results in sympatho-adrenal stimulation with an increase in circulating catecholamine concentrations, tachycardia and vasoconstriction.[4,5] Hypotension also stimulates the release of renin from the juxtaglomerular apparatus and this results in the activation of the potent vasoconstrictors angiotensin I and II.[6] Studies by Khambatta and colleagues[4,8] and Fahmy[7] have indicated that pre-operative beta-adrenoreceptor blockade with propranolol can modify the increases in catecholamines and plasma renin activity normally seen with hypotension induced by sodium nitroprusside.

Metoprolol was selected for groups 1 and 2 in this study because of its specificity (β_1), rapid onset of effect and brevity of action (plasma half-life 3 hours).[9] Oxprenolol was used in group 3 as an example of a beta-adrenoreceptor antagonist with some intrinsic sympathomimetic activity and a short duration of action (plasma half-life 2 hours).[9] Although there was a significantly lower requirement for nitroprusside in groups 1 and 3, it should be noted that the maximum individual infusion rate in any control patient was 4.2 μg/kg/minute. This is well within the upper limit of the recommended dose range (10 μg/kg/minute).[10,11]

The incidence of severe bradycardia, in five patients out of 30 who received a beta-adrenoreceptor antagonist, has not been reported previously, but the patients in the study by Khambatta and colleagues were premedicated with atropine, whereas ours were premedicated with diazepam. Also, their patients were drawn from a population with intracranial pathology, which is known to be associated with high circulating catecholamine concentrations. The anaesthetic technique in the study reported by Fahmy[7] differed from ours in that propranolol was used as the beta-adrenoreceptor antagonist, tubocurarine was used to enable controlled ventilation of the lungs. This may have resulted in administration of a lower concentration of halothane.

The haemodynamic response to induction was unexpectedly dramatic in view of the study patients' good general health and the relatively low doses of beta-adrenoreceptor antagonist which were used. This contrasts with the behaviour of the increasing number of patients who present for anaesthesia on much larger doses of these drugs. This may be because chronic therapy results in the development of compensating circulatory adjustments, such as an increase in the number of beta-adrenoreceptors as suggested by Boudoulas *et al.*[12]

We conclude that neither the potential improvement in operating conditions nor the reduction in sodium nitroprusside requirement, is an adequate compensation for the high incidence of profound bradycardia associated with acute pre-operative beta-adrenoreceptor antagonism with metoprolol or oxprenolol. In the absence of criteria which accurately predict the small group of patients who resist induced hypotension, the routine use of these drugs is not justified.

Acknowledgments

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Anaesthesia for combined heart and lung transplantation

J. P. SALE, D. PATEL, B. DUNCAN AND J.H. WATERS

Summary

In December 1983, Harefield Hospital started a programme for combined heart and lung transplantation. Fifty-two transplants have been successfully performed, with 36 survivors (April 1986). Patient selection and anaesthetic management of both donor and recipient are discussed. Two case histories are presented which illustrate the principles of anaesthetic management and postoperative care.

Key words

Anaesthetic techniques; heart–lung transplantation.
Surgery; cardiac, transplantation.

Organ transplantation over the last two decades has progressed enormously and kidney transplantation is now an accepted and highly successful therapy for end-stage renal disease. Recently, cardiac transplantation has become established as definitive therapy in selected patients. Isolated lung transplantation has yielded disappointing results with only 2 out of 28 patients surviving more than one month, and has now been abandoned in most centres. The main problems in isolated lung transplantation have been infection, failure of bronchial anastomotic healing and ventilation/perfusion mismatch. However, the results from combined heart and lung transplantation appear encouraging, although the long-term therapeutic value has still to be assessed. The advantages of combined heart/lung transplantation over simple lung transplantation are: firstly, there is better tracheal healing, as the donor trachea and lungs in a heart/lung transplant are adequately supplied by blood from the right coronary artery, unlike the lung transplant, where there is no bronchial blood supply; secondly, since all the diseased lung tissue is removed, ventilation/perfusion defects and the risks of recurrent infection are minimised.

Experimental work, pioneered in the 1950s,[1] has confirmed the case for continued heart and lung transplantation and that primates can withstand cardiopulmonary denervation. The first recorded heart/lung transplant in man was performed by Cooley[2] in September 1968; unfortunately the patient did not survive. No more attempts in man were made until cardiac transplantation was thoroughly established and until the advent of cyclosporin A for immunosuppression.[3] Shumway at Stanford started a programme of heart/lung transplantation in 1981,[4] when the first successful transplant was performed; the recipient was a 45-year-old woman with primary pulmonary hypertension, who is alive and well 4 years later.

In December 1983, Mr Magdi Yacoub started

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a programme of heart/lung transplantation at Harefield Hospital with encouraging results. Such a procedure is of particular interest to the anaesthetist and poses several challenging problems, which may influence both the early and late recovery of these patients. This article describes the anaesthetic management of patients for heart/lung transplantation at Harefield Hospital, and is illustrated with the presentation of two case histories.

Harefield Hospital has performed 52 heart/lung transplants, 36 of whom are alive, with the longest survivor being well at 20 months (at April 1986). Table 1 shows detailed patient data, with survival times and the original pathology for the first 20 patients.

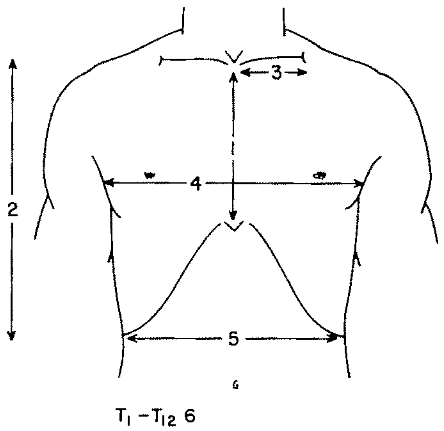

Fig. 1. Illustration of measurements made to assess and match size of donor to recipient. 1, Renal notch to xiphisternum; 2, acromion to lower rib margin; 3, sternal notch to acromion; 4, widest circumference at level of nipples; 5, circumference at lower rib margin; 6, length of vertebral spines of T_1–T_{12}.

Selection and management of donor

Careful selection and proper management of a suitable donor are vitally important for the successful outcome for the recipient. The potential donor should be young and without any cardiac or pulmonary pathology. If the death of the donor was caused by trauma of any sort there should obviously be no associated damage to the lung tissue. Good pulmonary function with satisfactory gas exchange with 30–35% oxygen and normal compliance are essential. Matching the size of the thoracic cavities of the donor and the potential recipient, as well as tissue compatibility, has also to be taken into consideration. Figure 1 illustrates the measurements taken to assess the size of the lungs.

Blood group ABO compatibility is necessary and tissue typing of the donor is obtained. Direct lymphocyte cross-match is carried out

Table 1. Patients who have received combined heart and lung transplantation: survival in days and pre-operative diagnosis of the first 20 patients, as completed up to August 1985.

Patient number	Sex	Age	Diagnosis	Length of follow-up (days)	Cause of death
1	M	32	Primary pulmonary hypertension	(10)	Multiple system failure
2	F	48	Thrombo-embolic disease	(19)	Multiple system failure
3	F	14	Primary pulmonary hypertension	362	A/W
4	F	36	Right lung disease	(57)	Infection
5	M	23	Cystic fibrosis	(46)	Infection
6	F	21	Primary pulmonary hypertension	315	A/W
7	M	39	Emphysema	291	A/W
8	M	30	Primary pulmonary hypertension	264	A/W
9	M	20	Eisenmenger's syndrome (single ventricle)	188	A/W
10	F	5	Pulmonary atresia	180	A/W
11	M	53	Eisenmenger's syndrome (VSD)	(165)	Rejection
12	F	43	Eisenmenger's syndrome (VSD)	79	A/W
13	M	46	Primary pulmonary hypertension	75	A/W
14	F	22	Eisenmenger's syndrome	(35)	Mutiple system failure
15	F	19	Eisenmenger's syndrome (VSD)	49	A/W
16	F	29	Eisenmenger's syndrome (VSD)	34	A/W
17	F	24	Interstitial lung disease post pneumonia	33	A/W
18	M	31	Primary pulmonary hypertension	32	A/W
19	F	42	Emphysema	19	A/W
20	M	3	Double outlet left ventricle	18	A/W

prior to transplantation; a negative cross-match of the recipient's serum against the donor's lymphocytes is essential. The safe cold ischaemia time for the myocardium has been well established, although this is not the case for lung tissue. In order to minimise ischaemic damage to the lungs, the donor is put on cardiopulmonary bypass (CPBP) in a routine manner and cooled to a core temperature of 8°C. During this period the lungs are ventilated with air at a rate of 5 breaths/minute to prevent alveolar collapse. The donor heart and lungs are then removed *en bloc.*

Early in the programme this was achieved by transferring the donor to Harefield Hospital, the donor heart and lungs being then harvested from an adjacent operating theatre to minimise ischaemia time. Later in the programme, this technique was successfully employed to harvest the organs from the donating hospital. The ischaemia time has been prolonged considerably, but this has not led to any detrimental lung function in the postoperative period. However, it has led to a significant increase in the availability of organs since, early in the programme, the understandable reluctance of the relatives to allow the transport of their brain-dead relative to another hospital had meant that few hearts and lungs were available.

Selection of recipient

Patients who may benefit from combined heart and lung transplantation can be divided into two main categories: those with severe irreversible pulmonary vascular disease and those with severe parenchymal lung disease (Table 2). Recipient patients should be those with an end-stage disease and severely symptomatic, where future deterioration and poor prognosis balance the risks of such a procedure. Current systemic infection and malignancy are absolute contraindications. Cyclosporin A, an immunosuppressant, may cause impairment of renal and hepatic function and it is preferable that there is no significant dysfunction of these organs. Unlike some other centres, this unit does not consider previous extensive cardiac or thoracic surgery as absolute contraindications to heart/lung transplantation. Similarly, there are no strictly defined age limits; the ages of our 20 patients have ranged from 3 to 53 years. It is also important that the patient be psychologically stable with a supportive family and be able to follow a complex postoperative medical regimen.

Table 2. Classification of pathology of those patients who may benefit from combined heart/lung transplantation.

A. *Pulmonary vascular disease*
- Primary pulmonary hypertension
- Eisenmenger's syndrome
- Thrombo-embolic disease
- Anatomical developmental anomalies
  (e.g. complex pulmonary atresia)
- Veno-occlusive disease

B. *Parenchymal lung disease*
- Obstructive disease
  (e.g. emphysema)
- Restrictive disease
  (e.g. cryptogenic fibrosing alveolitis)
- Destructive disease
  (e.g. cystic fibrosis)

Anaesthetic management

Pre-operative considerations

Heart and lung transplantation is a planned procedure dependent on the achievement of the best condition of the donor. However, one of the disadvantages of distant procurement is the difficulty in achieving this in the limited time available. Compatibility of the donor and recipient is assessed using ABO blood group, HLA histocompatibility and the presence or absence of cytotoxic antibodies. The cardiopulmonary status of the recipient is usually severely compromised and, as a consequence, there is a high intra-operative risk.

The patient is prepared for the theatre with an antiseptic bath and is reverse barrier nursed from this time. The immunosuppressive regimen of oral cyclosporin A (10 mg/kg) and azathioprine (2 mg/kg) and prophylactic antibiotics (ceftazidime and flucloxacillin) are started with the premedication.

The type and dose of premedication have to be judged carefully in the presence of severely elevated pulmonary vascular resistance and a fixed, low cardiac output. However, patients with primary pulmonary hypertension and Eisenmenger's syndrome can tolerate a relatively heavy premedication which is useful in minimising the dose of induction agents with their accompanying cardiac depression. For adult patients, we use oral diazepam (5–15 mg) followed, if necessary, by a suitable dose of intramuscular papaveretum and hyoscine. For our paediatric patients, we have used oral trimeprazine (2 mg/kg) followed by intramuscular papaveretum and hyoscine (3 mg/kg) with satisfactory results.

Infection, particularly pulmonary infection, is a major risk and a sterile ventilator with bacterial filters in the system is set up in the recipient's anaesthetic room. All anaesthetic equipment and drugs are prepared using the aseptic technique as described for heart transplantation by Grebenik and Robinson.[5]

Anaesthetic induction and intra-operative management

The anaesthetists are scrubbed and gowned and remain aseptic as far as possible throughout the induction of anaesthesia and the operative period. In the anaesthetic room, peripheral venous and radial arterial cannulae are inserted under local anaesthesia, and the arterial blood pressure is monitored continuously during induction.

Because of high, fixed pulmonary vascular resistance and a low cardiac output, a slow and smooth induction is essential. We have achieved this successfully in adults by using incremental doses of Diazemuls and phenoperidine followed by a suitable (small) dose of etomidate; pancuronium in a dose of 0.15 mg/kg is used to achieve neuromuscular blockade. Intubation of the trachea is performed using a sterile Portex orotracheal tube which is usually cut about 1–2 cm shorter in length to prevent the tip of the tube sitting at the tracheal anastomosis. Extreme care is taken to avoid hypoxia and hypercarbia throughout the procedure.

For our paediatric patients we have employed an inhalational technique using nitrous oxide, oxygen and halothane or intramuscular ketamine and a nitrous oxide–oxygen mixture. In Eisenmenger's syndrome, inhalational agents may cause a decrease in peripheral vascular resistance and hence an increased right to left shunt as well as direct myocardial depression. Hence these agents are discontinued as soon as peripheral venous access is established, and an intravenous anaesthetic agent substituted. Intermittent positive pressure ventilation not only reduces venous return, but also increases the pulmonary vascular resistance, which results in hypotension, increasing right to left shunt and increasing cyanosis. Hence, relatively small tidal volumes of 5–6 ml/kg are employed and the minute volume is adjusted to maintain normocarbia.

Despite careful induction, it is not unusual for patients to have a marked decrease in arterial blood pressure with increasing cyanosis. In our experience, some have responded to ventilation with 100% oxygen and a slow intravenous infusion of aminophylline which has both an inotropic and a pulmonary vasodilating effect. However, a variety of inotropic drugs have had to be employed to maintain a reasonable blood pressure, before the establishment of cardiopulmonary bypass.

Once a successful induction is achieved, a minimum of two lines are inserted in the left internal jugular vein (leaving the right side of the neck free for future cardiac biopsies) and after the bladder is catheterised the patient is ready for theatre.

Cardiopulmonary bypass is established in the usual manner with the aortic cannula placed high in the ascending aorta to allow access to the trachea. The patient is cooled to 25°C on bypass, using a mixture of crystalloid and colloid prime. The aorta is clamped and cardectomy performed. The lungs are then removed separately with the bronchi clamped to prevent soiling of the surgical field. Only the posterior pericardium and a cuff of the right atrium with the venae cavae attached, are left in the thorax.

Great care is taken to identify and preserve the phrenic, the recurrent laryngeal and the vagus nerves during the course of the dissection (damage to these nerves would have an obvious influence on the postoperative recovery of the patient). Once the thorax is eviscerated, meticulous care is taken to achieve haemostasis in the posterior mediastinum, since this site becomes virtually inaccessible once the donor heart and lungs are sutured in place. This is of particular relevance in patients with Eisenmenger's syndrome and other complex congenital heart defects who have extensive and large, thin-walled, collateral blood vessels.

The donor heart and lungs are placed anatomically *en bloc* into the thorax, and the trachea, aorta and the right atrium anastomosed in that order. Saline lavage and suction of the main bronchi of the donor lungs are performed prior to tracheal anastomosis just above the carina. The trachea is gently suctioned through the tracheal tube after completion of the anastomosis and at regular intervals thereafter using a strictly aseptic technique. The lungs are fully reinflated and, after testing the anastomosis for air leaks with an airway pressure of approximately 4.0 kPa, intermittent positive pressure ventilation (IPPV) is commenced using an air–oxygen mixture. A positive end expiratory pressure (PEEP) of 0.5–1.0 kPa is usually employed to prevent pulmonary atelectasis.

The $F\text{IO}_2$ is kept at or below 0.5 as the transplanted lungs may be more prone to oxygen toxicity. Methylprednisolone, 15 mg/kg, is given as aortic anastomosis is completed. After the aortic clamp is released to reperfuse the donor heart and lungs, a low-dose dopamine infusion (5 μg/kg) is commenced and the patient rewarmed to 37°C while the right atrial anastomosis is completed.

Ischaemic time for the donor organs is usually under 40 minutes when the donor is prepared in the adjacent theatre. Ischaemic times of over 3 hours have yielded successful results in lungs which have been procured outside Harefield Hospital. With revascularisation and rewarming, the donor heart spontaneously fibrillates, requiring electrical defibrillation. With the reestablishment of sinus rhythm, normothermia and normal acid-base status, the patient is taken off bypass. Before coming off bypass the trachea is once again carefully suctioned with a soft rubber catheter and then again at regular intervals to remove any blood and blood clots, since bleeding continues at the site of the tracheal anastomosis even after the administration of protamine. Aminophylline is used to improve pulmonary expansion and oxygenation. Blood and blood products are given as necessary; all the stored blood is filtered with 20-mm blood filters. Mannitol or frusemide are administered to maintain a good urine output throughout the procedure. Potassium supplements are avoided intra-operatively even if the plasma potassium level is below 3.5 mmol/litre. The donor heart seems to be particularly sensitive to potassium infusion which may precipitate cardiac dysrhythmias in the immediate post-pump period.

Postoperative management

Postoperatively the patient is barrier nursed in an isolation room and electively ventilated with a sterile ventilator and humidified, filtered gases. A positive end expiratory pressure (PEEP) of 0.5–1.0 kPa is employed until gaseous exchange is adequate with an $F\text{IO}_2$ of 0.5 or less. Care is taken to maintain a clear airway, and initially frequent tracheal suction is necessary to prevent obstruction of the tracheal tube with blood clots; scrupulous aseptic technique is especially important. Filtered blood is transfused to replace any losses, maintaining the haematocrit between 35 and 40%. Plasma is routinely used to maintain the oncotic pressure and the vascular volume. Crystalloids are initially kept to a minimum in order to reduce formation of pulmonary oedema and dextrose/saline solution is infused at a rate of 0.5 ml/kg/hour for the first 12 hours and at a rate of 1 ml/kg/hour thereafter. Potassium supplements are gradually introduced to replace losses (bolus doses over a short period are avoided). Urine output of at least 0.5 ml/kg/hour is maintained if necessary by using diuretics; we commonly use a mixture of frusemide and aminophylline for this purpose.

Extubation of the trachea of these patients follows the same criteria as for any major cardiac operation and is usually achieved within the first 18 hours. On disconnexion from the ventilator, spontaneous ventilation is usually established without any major problems although abnormal patterns of breathing have been noted. The cough reflex is present in the remainder of the recipient's trachea but absent in the transplanted lungs and therefore vigorous and regular chest physiotherapy is an important aspect of postoperative management. All the patients are also taught to do their own postural drainage. The indwelling cannulae are removed as soon as feasible and active mobilisation encouraged on the 3rd or 4th postoperative day. Antibiotics are continued for one week postoperatively and until all indwelling cannulae are removed. Antiviral and antifungal agents, acyclovir and nystatin, are started postoperatively and continued for 3 months; acyclovir may be continued for life if there is any evidence of viral infection. Immunosuppression at Harefield Hospital is based on a regimen of cyclosporin A and azathioprine, which is continued for life. Lung rejection presents a formidable diagnostic challenge and it is beyond the scope of this article to cover this complex subject. Briefly, lung rejection is monitored by serial chest X rays, combined with changes in clinical condition and gas exchange. Cardiac biopsies are performed as dictated by the clinical condition. Any evidence of rejection is treated with a course of methylprednisolone and/or antithymocyte globulins.

Case histories

Case 1

The first patient was a 14-year-old schoolgirl diagnosed as having primary pulmonary hypertension. Symptoms of shortness of breath on exercise were first noticed when she was 6 years old. She remained reasonably well until 1981, when her condition suddenly deteriorated; she became less tolerant to effort and had episodes of cyanosis. Soon she was unable to walk more than 100 metres without becoming extremely short of breath. There were no other symptoms.

On admission she had signs of right heart failure, with elevated venous pressure, enlarged liver and a marked precordial heave. Her electrocardiogram showed right axis deviation, together with right ventricular hypertrophy. Chest X ray showed marked cardiomegaly and prominent pulmonary arteries (Fig. 2). The pulmonary artery pressure was elevated, with a mean of 30 mmHg. Her cardiac output was so low that accurate measurements could not be determined and pulmonary vascular resistance could not be calculated. However, pulmonary function tests were essentially normal, as were resting blood gas results. She was not dyspnoeic

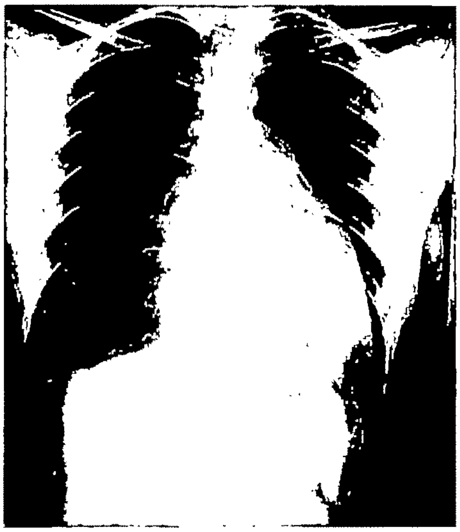

Fig. 2. Pre-operative chest X ray of case 1. This picture shows clear lung fields, and a massively dilated heart as a result of primary pulmonary hypertension and right heart failure.

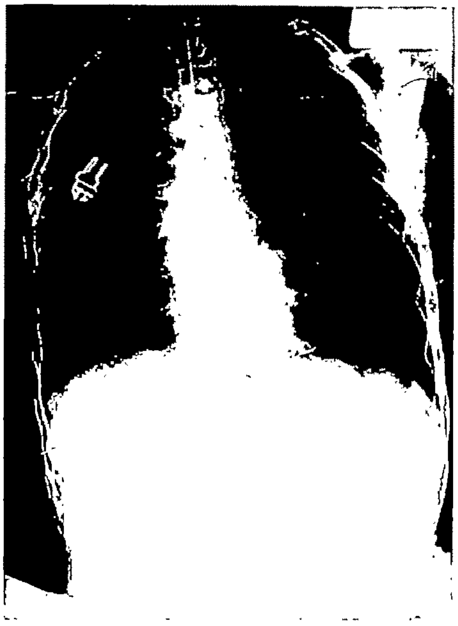

Fig. 3. A normal chest X ray of the donor for case 1, with a tracheal tube *in situ*.

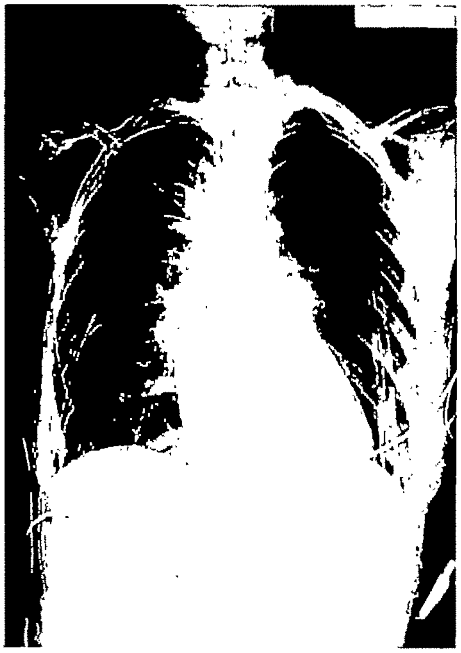

Fig. 4. Case 1: chest X ray taken immediately post-operatively. This is an essentially normal X ray of the transplanted heart and lungs, with bilateral chest drains and the tracheal tube *in situ*.

at rest nor dependent on oxygen. She presented for surgery and received the heart and lungs from a 14-year-old road traffic accident victim (Fig. 3).

Induction of anaesthesia was complicated by bradycardia and cyanosis, which were treated with atropine and aminophylline. Anaesthesia and surgery were otherwise uncomplicated; the whole procedure took about 6 hours. Following satisfactory cardiovascular stability and lung expansion (Fig. 4), the patient was extubated after 13 hours of elective ventilation. She resumed breathing without difficulty and maintained adequate gas exchange (Table 3). Her second postoperative day was characterised by a slow, deep pattern of respiration (8 breaths/minute). Although initially she had difficulty coughing and required regular physiotherapy, she made good progress and was mobilised at 3 days and walking by herself at 1 week. She had two episodes of mild lung rejection at 1 and 3 weeks (Fig. 5) which were successfully treated and she was discharged from hospital at 6 weeks.

Table 3. Post-transplantation arterial blood gas results (case 1).

	14 hours*	25 hours	Day 5	Day 8
$F\text{IO}_2$	0.5	0.4	Air	0.35
pH	7.39	7.48	7.53	7.52
$P\text{aO}_2$ (kPa)	20.76	25.0	8.8	8.2
$P\text{aCO}_2$ (kPa)	6.19	4.91	4.3	4.7
Respiratory rate (breaths/minute)	14	8	20	22

* Time of extubation.

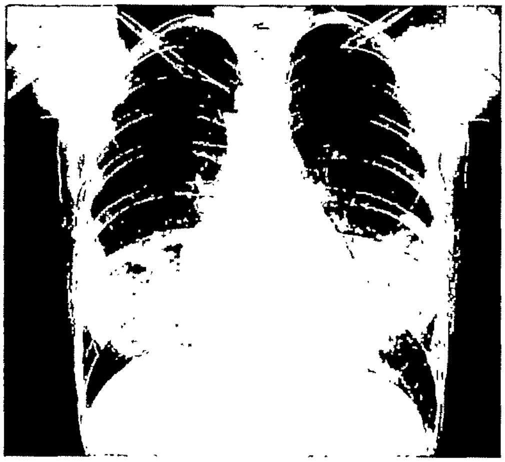

Fig. 5. Case 1: chest X ray showing an episode of acute rejection, at 3 weeks. Note the fine bilateral interstitial shadowing.

Case 2

The second patient was a 23-year-old male who was diagnosed as having cystic fibrosis soon after birth. Although he had all the manifestations of the disease with frequent chest infections, he remained reasonably well until a year before his operation. He then presented with cor pulmonale which was treated with digoxin and diuretics. His condition deteriorated, with increasing shortness of breath; an increasing frequency of chest infections produced copious amounts of purulent sputum and he needed oxygen at home.

Pre-operatively he was cachectic, and weighed only 44 kg. He had minimal signs of right heart failure, although he was grossly clubbed, and with a barrel-shaped chest. His chest X ray demonstrated bilateral patchy shadowing (Fig. 6) and an electrocardiogram showed right ventricular hypertrophy and strain. At cardiac catheterisation his pulmonary artery pressure was 65/25 mmHg, with a mean of 40 mmHg. Pulmonary vascular resistance was 6 Wood

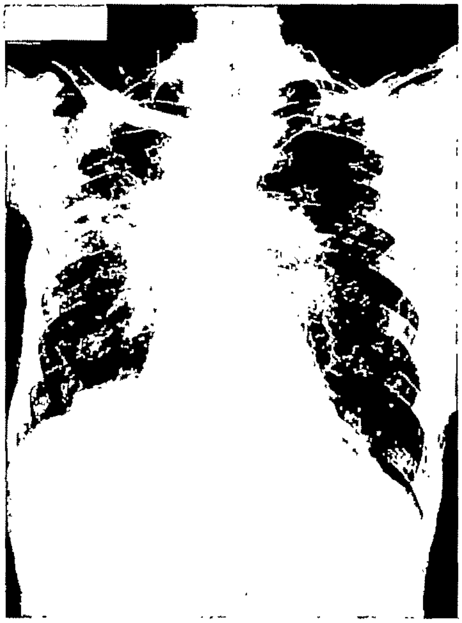

Fig. 6. Pre-operative chest X ray of case 2, showing all the pulmonary change of severe cystic fibrosis.

units* and all his lung function tests were abnormally low, with low lung volumes, reduced flow rates and low transfer factors. His respiratory rate pre-operatively was 60 breaths/minute and his arterial blood showed hypoxia ($P\text{ao}_2$ 6.4 kPa) and carbon dioxide insensitivity ($P\text{aco}_2$ 8 kPa; standard bicarbonate 31.5 mmol/litre). Sputum cultures demonstrated a heavy infection of *Pseudomonas* and *Staphylococcus aureus*.

He received the heart and lungs of a 22-year-old male who suffered a fatal head injury. Ischaemia time for the organs was 40 minutes. The operation took 8 hours and was complicated by massive haemorrhage from adhesions to the pleural cavity. He required 16 units of blood, but was finally transferred to the recovery room where he remained stable. He was extubated after 16 hours, and established spontaneous breathing without any problem. Figure 7 shows an immediate postoperative chest X ray. Initially he received 40% oxygen by facemask and his resultant arterial $P\text{ao}_2$ was over 20 kPa. Over the next few hours his respiratory rate decreased and his $P\text{aco}_2$ increased to 11.1 kPa with a standard bicarbonate of 42 mmol/litre, although he remained clinically very well. It was only after he was allowed to breathe room air that the trend reversed itself (Table 4).

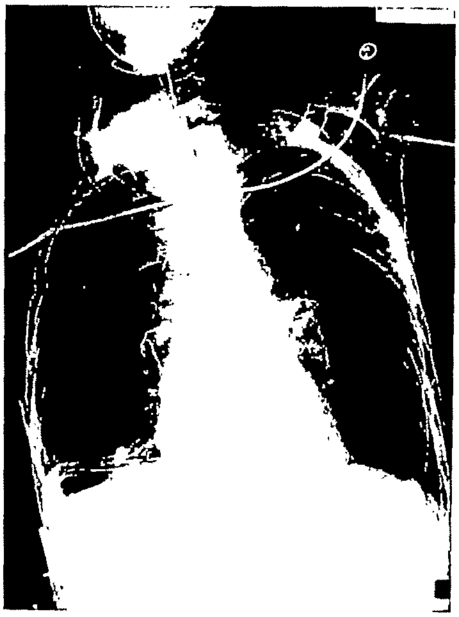

Fig. 7. Case 2: immediate postoperative chest X ray. Lung fields are clear and there are bilateral chest drains and a tracheal tube.

He made rapid recovery and was mobilised within a week. He continued to make good progress, having three episodes of mild rejection, and was discharged home after 5 weeks. However, within a week he was readmitted with severe shortness of breath and cyanosis as a result of bilateral chest infection with *Aspergillus fumigatus*. Despite aggressive treatment with antibiotics and physiotherapy, his condition deteriorated rapidly over the next week, and he required artificial ventilation with increasingly high minute volumes and inflation pressures. He died on the 46th postoperative day and the postmortem showed extensive and severe lung damage with *Aspergillus fumigatus*.

Table 4. Post-transplantation arterial blood gas results (case 2).

	Immediate	1 hour	2 hours	6 hours	24 hours	48 hours	56 hours	Day 7
$F\text{io}_2$	1.0	0.5	0.5	0.4	0.4	0.35	air	air
pH	7.38	7.39	7.50	7.45	7.39	7.41	7.43	7.47
$P\text{ao}_2$ (kPa)	5.15	10.6	18.08	26.3	24.6	20.2	9.9	8.8
$P\text{aco}_2$ (kPa)	5.25	6.8	4.58	4.94	8.44	11.1	7.6	6.3
Standard bicarbonate (mmol/litre)	23.4	28.5	27.4	26.5	33.9	42.1	37.3	33.5
Respiratory rate (breaths/minute)	IPPV	IPPV	IPPV	IPPV	14	20	22	16

Extubation at 16 hours.

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* Pulmonary vascular resistance in Wood units =

$$\frac{\text{mean left atrial pressure (mmHg)} - \text{mean pulmonary artery pressure (mmHg)}}{\text{cardiac output (litres)}}$$

Discussion

Combined heart/lung transplantation is still in its infancy. However, considering the enormity of such a procedure and especially the delicate nature of the organs concerned, the results from these initial cases are encouraging. Without doubt, this form of therapy offers the only hope for long-term survival in selected patients. For the anaesthetist such a procedure poses an exciting challenge and from the preceding case reports there are several points of interest.

Some recipients have poor oxygenation following re-inflation of the donor lungs. This may be due to atelectasis and oedema formation within the alveoli as a result of ischaemia during transplantation. There are no data on the viability of human lung following prolonged ischaemia, though donor heart is known to be viable for up to 4–6 hours. Animal lungs have been shown to tolerate ischaemia for up to 3 hours and the immediate response to short periods of ischaemia is pulmonary oedema, as a result of capillary leak and increased pulmonary vascular resistance. These changes have been shown to be readily reversible. This mechanism possibly explains the poor oxygenation immediately following re-inflation of the human donor lungs. In animals, prolonged ischaemia produces lung damage with desquamation of bronchial epithelium and the loss of mucosal function. There is also sloughing of pneumocytes and alveolar damage. These changes may reverse over 3 weeks, but subsequently there may be decreased ventilatory capacity and increased pulmonary vascular resistance.

In all our recipients the re-establishment of spontaneous breathing has been surprisingly uncomplicated. All the patients could breathe without difficulty and most had the power and the sensation to cough adequately. Neuronal control of breathing is via the respiratory centre in the brain stem, which is influenced both by peripheral and central chemoreceptor activity as well as by vagal afferents. Animal experiments have shown that the rhythmic discharge from the respiratory centre is spontaneous and is modified by the apneustic centre to produce inspiration, this being inhibited, in turn, by the pneumotaxic centre. Pulmonary stretch receptors, via the vagus nerve, also inhibit the apneustic centre. This is the Hering–Breuer reflex. In experimental models, dogs and cats rely on this reflex and bilateral denervation of the lungs produces an increased tidal volume and a slow respiratory rate with long periods of apnoea. This is referred to as apneustic respiration and is due to the unopposed inspiratory action of the apneustic centre. The exact role of the Hering–Breuer reflex in man still remains unclear, but it is obviously not essential for breathing. However, some of our patients have demonstrated an abnormal pattern and a slow rate of breathing, especially in the initial postoperative period, with subsequent reversion to normality.

The second case illustrates an interesting phenomenon. As a result of cystic fibrosis preoperatively he was a 'blue bloater' with carbon dioxide retention ($P\text{aco}_2$ 8 kPa). Replacement of his lungs removed the central hypoxic drive for respiration and resulted in carbon dioxide retention and the compensatory generation of bicarbonate; despite carbon dioxide concentrations of up to 11 kPa, he was fully alert and orientated with a normal rate and pattern of breathing. It was only after the patient was allowed to breathe room air that he became sufficiently hypoxic to reverse the process, and the bicarbonate returned to normal in about one week.

Unfortunately, we have little postoperative information, from our patients, about the long-term function of the transplanted lung. The Stanford group have followed a series of nine patients for 27 months.[6] They found that after operation all their patients showed a marked clinical improvement, with normal blood gases and transfer factors. Specific airway resistance and conductance were also normal. However, in the early transplant period, there was a marked decrease in most lung volumes that resulted in a restrictive ventilatory defect. Over the first year they found a steady improvement in lung function. These low lung volumes were thought to be due to disparity in volumes between the donor and the recipient, but they are also due to operative artefact, since any patient who has had cardiac or thoracic surgery shows a similar reduction in lung volumes in the early postoperative period.

Finally, lung rejection in heart/lung recipients can present a formidable diagnostic challenge. It is now clear that lung rejection can and does occur without cardiac involvement (and *vice versa*) and it can simulate a chest infection which can be life threatening in these patients. Lung rejection is monitored by daily clinical examination and serial chest X rays. Clinically it presents as pyrexia with tachypnoea and a cough, sometimes productive of watery secretions. There are also fine crepitations over both the lung fields and the blood gases show a sharp decrease in arterial oxygen tension and saturation. Radiologically, this is associated with a coarse granular shadowing (Fig. 5) which starts at the hilar regions and spreads to the base and the periphery. A course of antithymocyte globulin with or without methylprednisolone results in a dramatic improvement clinically (and of pulmonary gas exchange) but the radiological changes take a considerable time to show improvement.

In conclusion, combined heart/lung transplantation offers to selected patients symptomatic relief and the potential for a cure of their underlying cardiac and pulmonary pathology. However, such a procedure poses a number of questions. Of particular interest is the effect of denervation on such factors as control of respiration, airway reactivity and regulation of pulmonary blood flow. Similarly, the effects of transplantation on ciliary function and the metabolic function of the lung, and the effects of interruption of lymphatic and bronchial blood supply, are still unknown. These first patients have paved the way for future procedures which may provide answers to some of these questions.

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Anaesthesia and intra-ocular pressure in young children

A study of three different techniques of anaesthesia

G. DE L. DEAR, M. HAMMERTON, D. J. HATCH AND D. TAYLOR

Summary

This study compares the effects of three anaesthetic techniques on intra-ocular pressure (IOP) in infants and children: spontaneous ventilation using nitrous oxide with halothane (group 1) or isoflurane (group 2) and controlled ventilation with atracurium (group 3). The IOP remained constant in groups 1 and 2 until the administration of suxamethonium when it rose significantly; this rise was maintained during tracheal intubation. In group 3 IOP increased on administering atracurium and a further increase was seen during tracheal intubation. Diagnostic intra-ocular pressure measurements should be made during spontaneous breathing prior to tracheal intubation. The IOP was not directly affected by arterial blood pressure or by expired CO_2 tension.

Key words

Anaesthetics, volatile; halothane, isoflurane.
Complications; raised intra-ocular pressure.
Neuromuscular relaxants; atracurium.

Awake measurement of intra-ocular pressure (IOP) in children is difficult and so a general anaesthetic is often required. In diagnostic IOP measurements, for example in borderline glaucoma, it is essential that the anaesthetic does not significantly affect the IOP or prevent reliable assessment. Ketamine, for example, has been shown[1,2] to cause a rise in IOP in addition to its other adverse reactions.[3]

The purpose of this study was to compare the effects of three different anaesthetic techniques on IOP, two using inhalational agents and the third using controlled ventilation without volatile supplement. We also sought to determine the most appropriate moment to measure IOP during induction and maintenance of anaesthesia in order to obtain results which reflect awake IOP as closely as possible.

Patients and methods

The study was carried out in 60 infants and children between the ages of 10 weeks and 9 years who weighed between 4.5 and 40 kg, as shown in Table 1. The study was approved by the hospital ethical committee. A total of 101 eyes were studied, of which 74 were considered before the study to be normal and 27 abnormal, in which IOP was expected to be altered; for statistical purposes the two sections of the

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Table 1. Physical characteristics of the children.

Group	Age range (mean)	Weight (mean), kg
1	10 weeks–8 years (2.7)	4.8–24.8 (12.0)
2	6 months–9 years (3.5)	4.5–24.0 (12.8)
3	6 months–8 years (2.6)	4.7–40.0 (13.5)

Group 1, halothane; group 2, isoflurane; group 3, atracurium.

Table 2. Range of diagnoses in the two patient groups.

Normal pressure eyes (normal eyes)	Eyes with high pressure (abnormal eyes)
Blocked tear ducts	Congenital glaucoma
Congenital cataract	Retinoblastoma
Peter's anomaly*	Juvenile xanthogranuloma
Capillary haemangioma	Buphthalmos
Squint	Megalocornea
Steroid cataracts* (not on topical steroids)	Rhabdomyosarcoma
Bilaterial microcornea	
Retinal dysplasia	
Needling of posterior capsule	
Rieger's syndrome*	
Stickler's syndrome	
Coat's disease	

* Intra-ocular pressure previously found to be normal at examination under anaesthetic.

patient population studied were considered separately. Four patients also had awake measurements. The main ophthalmological anomalies are indicated in Table 2.

All patients were starved for 4–5 hours preoperatively and premedicated as follows: babies under 8 months of age were given atropine 0.02 mg/kg intramuscularly alone 45 minutes preoperatively; the remaining infants and children received premedication containing an opioid analgesic, and those below 15 kg received Inj. Peth Co. 0.07 ml/kg (1 ml contains pethidine 25 mg, promethazine 6.25 mg and chlorpromazine 6.25 mg) and atropine 0.02 mg/kg intramuscularly one hour before anaesthesia. Children weighing more than 15 kg received papaveretum 0.4 mg/kg and hyoscine 0.008 mg/kg intramuscularly 1.5 hours pre-operatively. The majority of patients were induced with cyclopropane and oxygen ($F\text{IO}_2$ 0.5), and with thiopentone 4–5 mg/kg in the six children over 7 years old.

Local anaesthetic (amethocaine HCl 1%) was applied to the conjunctiva as soon as the patient was asleep. General anaesthesia was maintained with N_2O and O_2 ($F\text{IO}_2$ 0.3) via an Ayre's T-Piece or a Bain system. The IOP measurements were made with a Perkins handheld applanation tonometer,[4] without using a speculum. All the IOP measurements were made by one investigator (M.H.). In all patients, the first IOP measurement (Ind) was made as soon as the Perkins tonometer was tolerated and within 3 minutes of the induction of anaesthesia.

The subjects were then randomly allocated into three groups of 20. In group 1 halothane was used as the volatile agent and a 2.5% concentration was added for 1.5 minutes after the Ind measurement. This was then reduced to 1.5% for a further 3 minutes, during which a vein was cannulated. The second IOP measurement (Inhal) was then made. Suxamethonium 1 mg/kg was given to facilitate laryngoscopy and intubation and the third reading (Relax) was made as soon as the patient was fully relaxed. The fourth reading (Tube) was made immediately after insertion of an oral tracheal tube. On resumption of spontaneous breathing, nitrous oxide, oxygen and halothane 1.5% were administered for 3 minutes and the fifth measurement (Stable 1) was made. The final IOP reading (Stable 2) was made after 3 further minutes at the same gas mixture.

In group 2, the protocol was similar except that isoflurane was substituted for halothane throughout. The initial concentration used was 4% for 1.5 minutes and then 2% for 3 minutes. After tracheal intubation the patients breathed 2% isoflurane for the last 6 minutes of the study.

In group 3, no volatile supplement was used and the only relaxant used was atracurium. Atracurium 0.5 mg/kg was injected immediately after the first IOP measurement (Ind) and the (Relax) reading made. Laryngoscopy and tracheal intubation took place 90 seconds after the administration of atracurium and the patient's lungs were ventilated with N_2O and O_2 alone. Efficacy of neuromuscular blockade was monitored with a Bard Peripheral Nerve Stimulator. The third measurement (Tube) was made immediately after tracheal intubation. A vein was then cannulated whilst the patient was ventilated manually for the remaining 6 minutes and final two measurements of the study.

Mean arterial blood pressure (MABP) and pulse rate were estimated using an automatic blood pressure monitor (Dinamap XT645) at the time of each IOP measurement. End-tidal expired carbon dioxide tension ($P\text{E}'\text{CO}_2$) was recorded concurrently using a paediatric cuvette (2 ml deadspace) connected to an infrared capnometer (Hewlett–Packard, Model 47210A) inserted in the system just proximal to the mask or tracheal tube. General anaesthesia was continued at the end of the study using either a spontaneous or controlled ventilation technique as appropriate to the surgical procedure, irrespective of grouping.

The standard error of the mean of differences from the induction (Ind) value was calculated; each patient acted as his own control. Significance at the 5% level was derived using Student's *t*-test.

Results

In four patients (six eyes) the IOP was measured under local anaesthesia pre-operatively and again throughout the study, and in two patients (four eyes) this procedure was repeated one week later. In these patients no significant difference was found between the pre-operative, post-induction or outpatient levels (Fig. 1). Each eye was used as its own control and all differences from the initial postinduction level were plotted (Figs 2 and 3, Table 3). The mean IOP after induction was 12 mmHg in the normal eyes and 22 mmHg in the abnormal eyes.

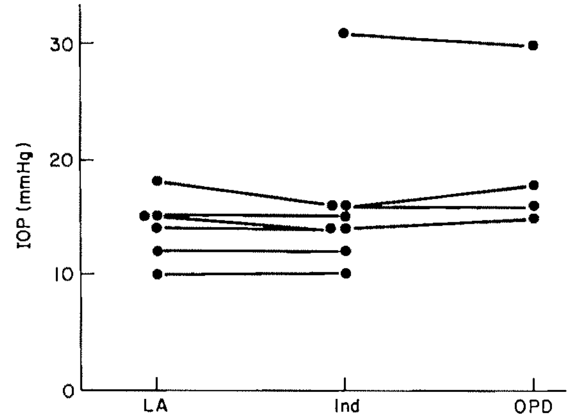

Fig. 1. Changes in intra-ocular pressure in patients assessed before (under local anaesthesia), during the study and afterwards in the outpatient department. IOP, Intra-ocular pressure (mmHg); LA, under local anaesthesia; Ind, after induction; OPD, in outpatient department.

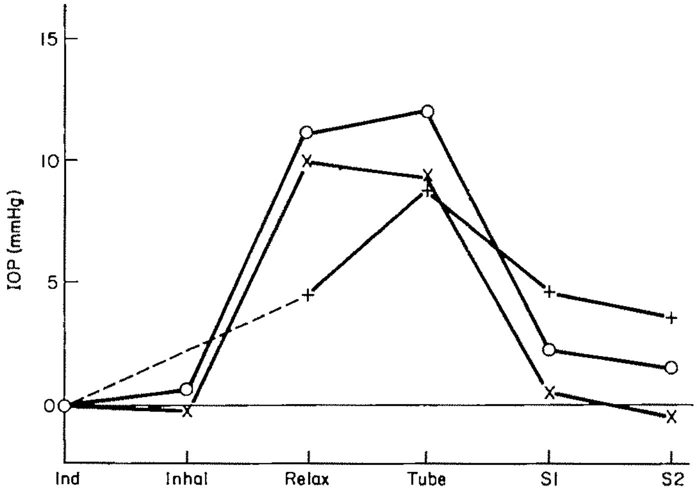

Fig. 2. Normal eyes: variation in intra-ocular pressure from mean of 12 mmHg at induction. IOP, Intra-ocular pressure (mmHg); Ind, induction; Inhal, addition of inhalational agent; Relax, addition of relaxant; Tube, after intubation; S1, Stable 1; S2, Stable 2. O, Halothane (group 1); ×, isoflurane (group 2); +, atracurium (group 3).

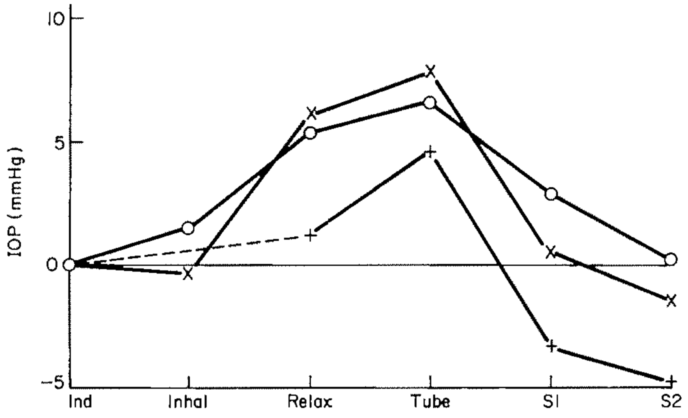

Fig. 3. Abnormal eyes: variation in intra-ocular pressure from mean of 22 mmHg at induction. ○, Halothane; ×, isoflurane; +, atracurium.

Table 3. Mean (SEM) changes in intra-ocular pressure (mmHg) from the induction recordings in the three groups at the times detailed.

Group	Inhal	Relax	Tube	Stable 1	Stable 2
Normal eyes					
1	0.7 (0.81)	11.2* (1.14)	12.0* (0.86)	2.3 (1.05)	1.6 (0.86)
2	−0.1 (0.9)	10.0* (1.18)	9.3* (1.18)	0.6 (0.76)	−0.5 (0.98)
3		4.4* (0.96)	9.0* (0.98)	4.7* (0.69)	3.4* (0.81)
Abnormal eyes					
1	1.4 (1.14)	5.3* (1.74)	6.7* (1.84)	2.8 (1.42)	0.2 (1.27)
2	−0.4 (1.23)	6.1* (0.73)	7.9* (1.42)	0.5 (0.79)	−1.4 (1.17)
3		1.2 (1.31)	4.7 (2.12)	−3.3 (1.59)	−4.7 (1.92)

Inhal, addition of inhalational agent; Relax, addition of relaxant; Tube, after intubation.
* Significant at $p<0.05$.

The addition of either halothane or isoflurane did not significantly alter the IOP in the children with normal eyes (Table 3). The administration of suxamethonium resulted in a significant increase in IOP in groups 1 and 2, and there was also a moderate but significant elevation of the pressure following the administration of atracurium. The increase in groups 1 and 2 was maintained at laryngoscopy and intubation but was increased in group 3. In all patients the IOP decreased after tracheal intubation. In both groups 1 and 2 this fall reached a pressure not significantly different from the induction (Ind) value, but in the atracurium group the IOP remained slightly elevated.

The initial IOP in the abnormal eyes was higher and did not alter as much as in the normal eyes. No significant rise was noted after the addition of the volatile agent but, as in the normal eyes, the IOP rose significantly after the administration of suxamethonium and increased still further after laryngoscopy. There was no significant change in IOP in group 3 after the addition of atracurium, in contrast to the normal eyes, where the increase was significant. In all three groups there was a return to the initial IOP during the stable period and, indeed, by Stable 2 in groups 2 and 3 the IOP was below the baseline pressure. In group 3 the fall was rapid after tracheal intubation and the IOP finished almost 5 mmHg lower.

The $P_{E'}\mathrm{CO}_2$ level in all three groups remained stable until after tracheal intubation (Fig. 4), when an increase was noted. In groups 1 and 2 the increase was maintained and in the isoflurane group $P_{E'}\mathrm{CO}_2$ rose slightly, to 6.6 kPa. In the atracurium group $P_{E'}\mathrm{CO}_2$ fell back to the baseline value during manually controlled venti-

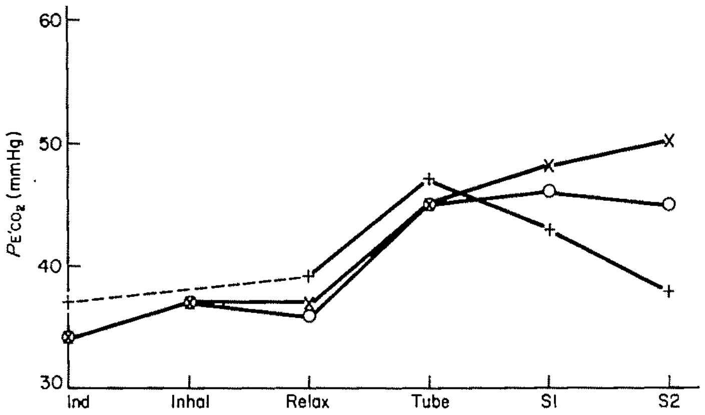

Fig. 4. Changes in expired carbon dioxide tension ($P_{E'}CO_2$) in the three groups throughout the study. ○, Halothane; ×, isoflurane; +, atracurium.

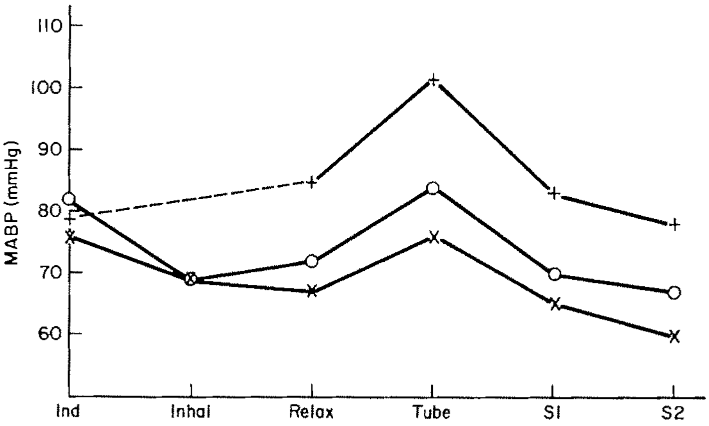

Fig. 5. Changes in mean arterial blood pressure (MABP) in the three groups. ○, Halothane; ×, isoflurane; +, atracurium.

lation. The mean arterial blood pressure, calculated from the systolic and diastolic pressures, decreased during the Inhal period and changed little after the administration of suxamethonium in groups 1 and 2. In group 3 the administration of atracurium had little effect on MABP. In all three groups tracheal intubation was associated with an increase in MABP which returned to or decreased below the baseline level during the Stable periods (Fig. 5).

Discussion

It is important that measurements under anaesthesia reflect awake levels for diagnostic IOP measurements in uncooperative children. This is especially true when the measurement of IOP is vital to management, as for example in a patient with borderline glaucoma. During and after therapy both reliable and repeatable measurements are necessary. This study shows the variation in IOP that occurs during anaesthesia produced by standard techniques.

Many procedures and drugs have been shown to affect IOP.[5] Two in particular which cause a rise are suxamethonium[6,7] and laryngoscopy/intubation;[8,9] the effect of these was confirmed in the present study (Figs 2 and 3, Table 3). Intravenous lignocaine has been shown to reduce the pressor response following intubation[10] and thus to minimise the rise in IOP, but we did not evaluate this effect in the present study. Many other drugs have a significant effect on IOP and most cause a decrease.[5] These include carbonic anhydrase inhibitors, osmotic diuretics, central nervous depressants such as tranquillisers, barbiturates, narcotics and neurolept agents. Both halothane and isoflurane have been shown after pre-operative sedation not to affect IOP,[11] although Earnshaw[14] has shown isoflurane to cause a fall in IOP. Ketamine is another agent with which conflicting results[1–3] have been shown and, in addition, many anaesthetists find its dissociative effects undesirable. No change in IOP has been reported after administration of anticholinergics.[13] It is well known that coughing, straining and vomiting[4,14] raise the IOP, and any children that were unduly distressed before anaesthesia started were excluded from the trial.

There was a significant increase in IOP after administration of suxamethonium in the present study. Laryngoscopy and intubation were associated with an increase in group 3, even when not preceded by suxamethonium. It is possible that this increase could have been caused by atracurium, though it seems surprising, if this were so, that the increase was not fully revealed in the measurement made 90 seconds after its administration (Relax), when peripheral nerve stimulation demonstrated a 90% block. This fact has implications in penetrating eye injuries, when a marked increase in pressure should be avoided and suxamethonium is still the best drug to facilitate rapid tracheal intubation; it has been suggested that giving lignocaine may prevent the increase in this case.

We found some practical disadvantages associated with the use of atracurium in our technique. It is preferable to use an intravenous line, which needs to be established either before induction, without upsetting the patient, or very soon afterwards. Awareness can be a problem if no volatile agent is used for a long time, especially if adequate premedication has not been given. In the patients who received controlled ventilation and atracurium with no volatile supplement (group 3), variation from the baseline IOP was significant and led to difficulty in interpretation of the changes in the pressure measurements.

No direct relationship was found between the IOP measurement and MABP[5] or $P\text{E}'\text{CO}_2$. The blood pressure increased at tracheal intubation but remained low in groups 1 and 2 after the administration of suxamethonium, when the IOP increased significantly. Similarly, $P\text{E}'\text{CO}_2$ was at its highest in the isoflurane group (group 2), but the IOP was low at Stable 2 (see Table 3 and Fig. 4). The reduction of up to 50% in the IOP of manually ventilated patients[15] was not seen in the atracurium group (group 3).

The wide variation in the levels recorded in the pathological eyes indicates that in these patients little can be concluded from the IOP measurement, especially after tracheal intubation. In the normal pressure eyes, the mean IOP was approximately half that found in the eyes with high pressure (12 mmHg against 22–24 mmHg), and the measurement did not significantly alter from baseline after the addition of the inhalational agent (Inhal), whether it was halothane or isoflurane. The end of the study, that is, at least 6 minutes after tracheal intubation, is probably not a good time for making diagnostic IOP measurements. The pressure was not different from baseline, but there may be patient-to-patient variation in the time taken for the elevating effects of the relaxant and laryngoscopy/intubation to wear off.

The choice of Ind, the period just after induction, as the baseline was vindicated in those patients in whom pre-operative, postoperative and outpatient measurements were identical (Fig. 1). The measurement of intra-ocular pressure in children just after induction requires close cooperation between anaesthetist and ophthalmic surgeon, as well as speed and accuracy. Both inappropriate techniques that give rise to pressure on the eyeball, such as Schiotz tonometry used by many workers,[1,6,8,12,13] or the use of a poorly fitting speculum,[16] may falsely increase IOP.

The development of a routine, preferably using the same team, for diagnostic measurement of IOP in infants and children is advised. The most suitable time to measure IOP was found to be just after induction and up to the administration of suxamethonium in those patients who breathed halothane or isoflurane spontaneously. Isoflurane produced the least change in the absolute level of IOP measurements and it may be appropriate to use this agent to avoid false readings unless measurement can be made early on.

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Anaesthesia, 1987, Volume 42, pages 266–269

A comparison between the effects of fentanyl, droperidol with fentanyl, and halothane anaesthesia on intra-ocular pressure in adults

R. O. FENECK AND M. A. DURKIN

Summary

The effects of fentanyl 3.0 μg/kg (group 1), droperidol 0.1 mg/kg with fentanyl 3.0 μg/kg (group 2), and halothane 0.5% inspired concentration (group 3) on intra-ocular pressure were compared. In each group, a decrease in intra-ocular pressure was produced which was significantly lower than resting values ($p > 0.01$) and was independent of changes in arterial blood pressure. The recovery time in group 1 patients was significantly less than that of patients in group 3.

Key words

Surgery; ophthalmic.
Measurement techniques; intra-ocular pressure.

General anaesthesia for intra-ocular surgery provides comfort and safety for the patient and a degree of control of factors that affect intra-ocular pressure (IOP) during surgery. Many of these factors have been described, i.e. arterial[1,2] and central venous pressure[3] and arterial carbon dioxide tension.[4,5] In this study, we have standardised the factors known to affect IOP, in order to separate the effects of the three anaesthetic techniques described. In addition, we have continued our study into the early post-operative period, in order to ascertain whether there are any differences in recovery between the three groups.

Methods

Thirty adult patients (14 males and 16 females) who presented for cataract extraction under general anaesthesia and who gave informed consent were studied. All patients received a standard oral benzodiazepine premedication 2 hours pre-operatively. The patients were randomly divided into three equal groups: group 1, fentanyl 3.0 μg/kg; group 2, droperidol 0.1 mg/kg with fentanyl 3.0 μg/kg; group 3, halothane 0.5% inspired concentration. The anaesthetic sequence was as follows.

Group 1

Intravenous fentanyl was given, followed by thiopentone to obtund the eyelash reflex, pancuronium 0.1 mg/kg and artificial ventilation of the lungs with 50% oxygen in nitrous oxide for 3 minutes, 100 mg topical lignocaine to the larynx, tracheal intubation and the establishment of intermittent positive pressure ventilation (IPPV) with 33% O_2 in N_2O. End tidal carbon dioxide concentration was monitored using an infrared analyser (Wilks Ltd) and kept constant at 5%.

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Group 2

Intravenous droperidol was given, followed by fentanyl in the doses described, thiopentone to obtund the eyelash reflex and then as above.

Group 3

Intravenous thiopentone was given to obtund the eyelash reflex, followed by pancuronium and artificial ventilation of the lungs as described above, but with 0.5% halothane added to the inspired gas mixture. Halothane was vaporized from a Fluotec Mk II vaporizer; this had been checked using an ultraviolet analyser.

All measurements were made with the patient supine and with no tilt on the operating table. Arterial blood pressure (BP), IOP and heart rate (HR) were recorded at the following times: before induction of anaesthesia; after stabilisation of anaesthesia, before surgery; after surgery, before cessation of anaesthesia and reversal of neuromuscular blockade.

Arterial blood pressure was measured with an oscillotonometer; heart rate was measured from the ECG (standard lead II). Intra-ocular pressure was measured using a Perkins hand-held applanation tonometer.[6] Following topical application of local anaesthetic and fluorescein, three readings were taken and the mean value recorded. All measurements were made in the eye not about to undergo surgery; no patient had had previous eye surgery. Following induction of anaesthesia, the right external jugular vein was cannulated and the pressure measured using a saline manometer. Recordings were made at points 2 and 3.

Immediately after the third series of measurements, anaesthesia was discontinued and neuromuscular blockade reversed with atropine 1.2 mg and neostigmine 2.5 mg intravenously. The time taken to establish spontaneous ventilation was recorded. The patients were transferred to the recovery room where they were observed by one of the authors. The time taken to recovery was determined by establishing when the patients were able to perform a simple task to command (squeezing the observer's hand) and when they were able to repeat their full name.

At 15, 30 and 60 minutes after the establishment of spontaneous ventilation, further recordings of arterial blood pressure, heart rate and respiratory rate were taken and the incidence of nausea or vomiting, analgesic and antiemetic requirements, as well as any untoward effects, noted. Intramuscular pethidine (0.75–1.0 mg/kg) was given for postoperative analgesia when required. Prochlorperazine (6.75–12.5 mg) was given for antiemesis when required. Analysis of data was carried out using the Chi squared test and paired and unpaired Student's *t*-test; a probability value of less than 5% was accepted as significant.

Results

The patients were comparable on the basis of body weight and resting levels of BP, HR and IOP. The duration of surgery was similar in each group (Table 1). Table 2 shows the changes in IOP which occurred in each group at the times stated. In each group, IOP was significantly reduced during anaesthesia when compared to resting levels. In each group, BP fell after induction of anaesthesia (Table 3); however,

Table 1. Analysis of pre-induction measurements; values expressed as mean (SEM).

	Group 1	Group 2	Group 3
Age, years	69.1	70.5	59.8
Weight, kg	68.1	67	73.4
	(3.8)	(5.1)	(3.2)
BP, mmHg	110	116	114
	(3.6)	(3.9)	(4.2)
HR, beats/minute	77	77	75
	(6.1)	(5.1)	(5.5)
IOP, mmHg	13.3	16.1	13.9
	(1.01)	(1.38)	(1.38)
Duration of surgery, minutes	59	61	64
	(3.1)	(7.1)	(5.3)

Table 2. Measurements of IOP (mmHg) in each group at the times shown; in each group, values marked with an asterisk are significantly different from pre-induction measurements. Values expressed as mean (SEM).

	1 Pre-induction	2 Before surgery	3 After surgery
Group 1	13.3	6.8*	8.5*
	(1.0)	(0.7)	(1.3)
Group 2	16.1	7.6*	9.1*
	(1.2)	(1.3)	(1.4)
Group 3	13.9	8.3*	8.9*
	(1.2)	(0.8)	(1.0)

*$p < 0.01$.

Table 3. Measurements of BP (mmHg) in each group at the times shown; in each group, values marked with an asterisk or dagger are significantly different from pre-induction measurements. Values expressed as mean (SEM).

	1 Pre-induction	2 Before surgery	3 After surgery
Group 1	111 (3.6)	91† (4.6)	105 (5.1)
Group 2	116 (3.5)	87† (7.9)	104 (5.3)
Group 3	114 (3.8)	91* (6.8)	99 (6.0)

* $p < 0.02$.
† $p < 0.01$.

comparison of individual measurements of BP and IOP throughout the study shows a poor correlation between the two parameters (*r* 0.2). Adequate spontaneous ventilation was easily achieved in each group following cessation of anaesthesia and reversal of neuromuscular blockade. However, the time from onset of spontaneous ventilation to time to obey commands was significantly longer in group 3 than in group 1 (Table 4).

Discussion

Pancuronium was used as the sole relaxant in this study, since others have found little change in IOP following its use.[7,8] We were happy to use right external jugular venous pressure as an estimate of CVP; the correlation between right internal and external jugular venous pressure in patients undergoing IPPV is described elsewhere.[9]

In each group of patients, anaesthesia wa associated with a reduction in BP and IOI However, the correlation between IOP and B in this study was not significant and, indeed, th poor relationship between levels of BP and IO within the physiological range is well estab lished.[10] Workers who have reported a co relation between arterial and ocular pressur have used halothane anaesthesia to produc arterial hypotension;[1] this agent is reported t produce ocular hypotension even when BP i well maintained.[11]

It is well established that anaesthetic tech niques that utilise halothane,[11] enflurane[12] an trichloroethylene,[13] all cause a reduction in IOI However, inhalational agents, particularly in a elderly group of patients (such as those reporte in this study), are known to have the potentia to cause cardiovascular depression, especially i the concentration of volatile agent is increase in an attempt to improve ocular conditions fo surgery.[14]

Normocapnic general anaesthesia using fent anyl, in a technique similar to that describe here, has been shown to lower IOP[15] and ha been recommended for use in elderly patient undergoing cataract extraction,[16] although dat that compare this technique with the use o volatile agents are not available. The maximur depression of IOP during fentanyl anaesthesi has been reported as occurring approximatel 10 minutes after its administration.[15] This interval between induction of anaesthesia and commencement of surgery was similar to that see in our study, although in routine clinical practic it may be that this interval would be furthe shortened.

The addition of droperidol to fentanyl ma

Table 4. Comparison of recovery from anaesthesia between the groups; values expressed as mean (SEM).

	Group 1	Group 2	Group 3
Time to onset of spontaneous ventilation, minutes	5.1 (1.3)	4.2 (0.9)	5.2 (0.7)
Time to obey commands, minutes	13.1 (2.2)	18.3 (2.6)	24.1* (2.2)
Number of patients who required analgesia during first 4 hours postoperatively	3	2	9†
Number of patients who required antiemesis during first 4 hours postoperatively	2	2	8†

* Group 3 significantly greater than group 1 ($p < 0.01$).
† Group 3 significantly greater than groups 1 and 2 ($p < 0.005$).

be thought to have certain specific advantages; in particular, the dangers of light anaesthesia in this group of patients (coughing, straining on the tracheal tube) would be reduced, as would the risk of awareness during anaesthesia. Droperidol is also a potent antiemetic and thus might be thought to be useful following ophthalmic surgery, since repetitive postoperative vomiting may raise intra-ocular pressure and thus threaten the eye, as well as being extremely distressing for the patient. In practice, we found little advantage in adding droperidol to fentanyl in this elderly group of patients. We observed no signs of unduly light anaesthesia in any of the patients in this study, and the requirements for postoperative analgesia and antiemesis were similar in groups 1 and 2; these were significantly less than both analgesic and antiemetic requirements for patients in group 3. However, it might be felt that this aspect of the study is unduly biased against those patients in group 3 who did not receive any conventional analgesic preoperatively.

Historically, ophthalmic surgery has been associated with postoperative regimens designed to produce profound sedation and thus to ensure minimal alteration of intra-ocular pressure, as might occur following change in posture, coughing and general early mobilisation. These measures were designed to give the eye the best chance of healing successfully. The development of controlled general anaesthesia and microsurgical techniques has meant that prolonged postoperative sedation is no longer necessary to protect the eye and, indeed, early recovery is preferred since it minimises the risk of developing bronchopneumonia, pulmonary embolus and other complications to which elderly patients are susceptible. We believe that in this study we have demonstrated an anaesthetic technique which combines good intra-operative conditions with early recovery.

In conclusion, we have shown that IOP is well controlled in this study in all three patient groups. However, the shorter recovery time in patients in group 1 (fentanyl) may be of benefit in promoting a safer postoperative recovery. We were unimpressed with the necessity for adding droperidol when using this technique and, indeed, we would prefer to add a small increment of volatile agent during surgery should this become necessary in order to further enhance intra-ocular conditions.

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We wish to thank the ophthalmic surgeons and anaesthetists at St. Thomas's Hospital for their advice and encouragement during this study.

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Vomiting after ophthalmic surgery

Effects of intra-operative antiemetics and postoperative oral fluid restriction

A. A. VAN DEN BERG, A. LAMBOURNE, N. S. YAZJI AND N. A. LAGHARI

Summary

The usefulness of intra-operative antiemetics and postoperative oral fluid restriction in the prevention of vomiting following anaesthesia for ophthalmic surgery, was studied in 200 patients. They were allocated into four groups of 50 and given either saline (as control), droperidol, metoclopramide or prochlorperazine. Oral intake was restricted postoperatively in half of the patients of each group. Anaesthesia comprised morphine and atropine premedication and a halothane, nitrous oxide and oxygen spontaneous breathing technique. No significant beneficial effects resulted from intra-operative antiemetics; vomiting incidences of 26% after saline and droperidol, 28% after metoclopramide and 14% after prochlorperazine were observed. Younger patients and females vomited most frequently. Restriction of oral fluids did not decrease the incidence of vomiting but demonstrated that approximately half of those patients who vomit do so with their first postoperative oral intake. Vomiting was observed more frequently after non intra-ocular surgery than after intra-ocular surgery (37% cf. 16%, $p < 0.01$) and postoperative analgesics were required by more non intra-ocular patients than by intra-ocular patients (25% cf. 5%, $p < 0.001$). Squint patients vomited most frequently (48%) and most frequently required postoperative analgesia (35%).

Key words

Vomiting; antiemetics, nausea.
Surgery; ophthalmic.

Postoperative vomiting following intra-ocular surgery is hazardous as it occasionally gives rise to ocular wound dehiscence, iris prolapse or intra-ocular bleeding. The problem of postoperative vomiting following anaesthesia for all types of surgery has always been of concern and a substantial literature has accrued identifying factors that contribute to its causation.[1] Ophthalmic surgery has the additional possibility of an emetic effect resulting from surgical manipulation of the eye. Studies aimed at reducing the incidence of postophthalmic surgical vomiting are therefore of special interest to ophthalmic anaesthetists.

The reported incidences of vomiting following ophthalmic surgery vary widely, from zero in male patients after all types of ocular surgery under local anaesthesia,[2] to 85% in children after squint correction under general anaesthesia.[3] Of the many antiemetic drugs, droperidol (half-life

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2.3 hours after intravenous injection,[4] with a dopaminergic receptor blocking action[5] in the chemoreceptor emetic trigger zone[6]) has been shown to be an effective antiemetic when given routinely either pre-, intra- or postoperatively for all types of ophthalmic surgery under both local and general anaesthesia.[2,3,7,8]

This prospective study was undertaken to compare two alternative popular antiemetic drugs, metoclopramide and prochlorperazine, with droperidol in the reduction of vomiting after eye surgery. Metoclopramide has both central and peripheral sites of action[9,10] and a half-life of 2.76 hours after intravenous injection,[11] whilst prochlorperazine, a phenothiazine congener with uncertain pharmacokinetics[12,13] (usually likened to those of chlorpromazine[14]), is said to have a half-life of 23 hours.[15]

Two observations were made as the study progressed. Firstly, emesis appeared frequently to follow postoperative oral fluid intake; the study was accordingly extended to assess the antiemetic effect of delaying the first postoperative oral intake. Secondly, emesis and pain appeared to be more frequent following non intra-ocular surgery. In view of the greater hazard from vomiting after intra-ocular surgery, data were further analysed after classifying patients according to the site of operation (intra-ocular or non intra-ocular).

Method

Two hundred consecutive patients were studied. Patients were randomly assigned to one of four drug groups so as to receive either saline 1–2 ml intravenously intra-operatively as control, droperidol 0.035 mg/kg intravenously with induction, metoclopramide 0.15 mg/kg intravenously at the end of anaesthesia, or prochlorperazine 0.18 mg/kg intramuscularly intra-operatively. The first 100 patients were allowed early postoperative oral fluids, whilst the second 100 patients were not allowed oral fluids postoperatively for at least 2 hours in neonates and children and for at least 4 hours in adults.

The same anaesthetic technique was used in all patients. Premedication consisted of morphine 0.1–0.15 mg/kg and atropine 0.008–0.01 mg/kg intramuscularly approximately one hour pre-operatively. Adults were pre-oxygenated whilst an intravenous infusion was established. Following alcuronium pretreatment (0.03 mg/kg) anaesthesia was induced with a sleep dose of thiopentone and tracheal intubation achieved after suxamethonium 1.0–1.5 mg/kg and spraying the larynx with lignocaine. Children were induced with halothane in nitrous oxide and oxygen; an intravenous infusion was then established and alcuronium pretreatment (0.03 mg/kg), thiopentone 3–4 mg/kg and suxamethonium 1.0–1.5 mg/kg were given to standardise the induction sequence.

Intra-operative intravenous infusions of Ringer's lactate, dextrose saline, normal saline, or 10% dextrose with added insulin and potassium in diabetic patients only, were continued until oral fluids were tolerated postoperatively. Anaesthesia was maintained with nitrous oxide, oxygen and halothane breathed spontaneously using either a circle absorber or a Jackson Rees system. The operating table was inclined 10–15° head up in all cases. Morphine, pethidine, paracetamol and metoclopramide were prescribed for postoperative analgesia, nausea and vomiting.

Postoperative oral fluids were started in the wards according to each patient's state of wakefulness. In the non fluid restricted group, those well awake were offered a sip (30 ml) of water hourly after 1–2 hours; drowsy patients were encouraged to take similar amounts after 3–4 hours and very drowsy or sleeping patients were allowed to rest until they awoke; fluids were then encouraged as above. The same approach was followed in the fluid restricted group, except that no oral fluids were allowed or offered during the first 2 hours in children and for the first 4 hours in adults.

Note was taken of pre-operative vomiting, surgery performed, duration and details of anaesthesia and duration of recovery ward time; the latter was used as an index of any sedative effect of the antiemetics. All patients were visited on the first postoperative day and record made of retching and vomiting, their time of occurrence and the incidence, dosage and time of postoperative analgesics and antiemetics. The relationship between vomiting and first oral intake was enquired into and the time of first postoperative oral intake obtained from fluid balance charts. Nursing staff were also asked to report any extrapyramidal reactions.

Analysis of variance was used to compare patient groups. Chi squared tests were used to test for significance in vomiting and analgesic requirement incidences. The effect of the anti-

Table 1. Intra-operative antiemetics. Comparison of patients in drug groups.

			Age (years)		Weight (kg)		Duration of anaesthesia (minutes)		Recovery ward time (minutes)	
Drug group	Sex	Number	Mean	Range	Mean	Range	Mean	Range	Mean	Range
	F	20	46.1	<1–72	49.7	4–79	81.7	45–140	38.2	20–75
Saline	M	30	47.3	<1–80	48.9	4–76	64.5	25–185	35.9	20–65
	Total	50	46.9	<1–80	49.2	4–79	71.4	25–185	36.7	20–75
	F	23	43.5	<1–76	50.1	5–94	67.8	30–164	38.0	25–160
Droperidol	M	27	47.4	<1–81	57.3	9–92	69.6	20–115	42.2	17–95
	Total	50	45.6	<1–81	54.0	5–94	68.8	20–160	40.2	17–95
	F	18	43.5	<1–83	45.5	4–84	76.2	30–190	44.6	5–180
Metoclopramide	M	32	41.3	<1–82	46.1	4–85	70.6	32–125	39.4	17–90
	Total	50	42.1	<1–83	45.9	4–85	72.7	30–190	41.1	5–180
	F	15	37.9	<1–74	43.5	5–79	65.1	20–105	40.2	15–110
Prochlorperazine	M	35	35.7	<1–79	44.9	5–85	65.5	20–115	37.1	20–85
	Total	50	36.4	<1–79	44.5	5–85	65.4	20–115	38.1	15–110
	F	76	42.5	<1–83	47.6	4–94	72.9	30–190	40.1	5–180
Overall	M	124	42.5	<1–82	48.9	5–92	67.5	20–185	38.5	17–95
	Total	200	42.5	<1–83	48.4	4–94	69.6	20–190	39.3	5–180

Table 2. Postoperative vomiting. Vomiting, sex and age analyses in drug groups and overall.

	Saline		Droperidol		Metoclopramide		Prochlorperazine		Overall	
Vomiting										
Patient numbers	50		50		50		50		200	
Number vomiting	13 (26%)		13 (26%)		14 (28%)		7 (14%)		47 (24%)	
Multiple vomiters	5 (38%)		3 (23%)		7 (50%)		3 (43%)		18 (38%)	
Single vomiters	8 (62%)		10 (77%)		7 (50%)		4 (57%)		29 (62%)	
Mean time of vomiting (hours)	4.9		4.8		3.5		7.6		4.4	
Sex analysis	M	F	M	F	M	F	M	F	M	F
Patient numbers	30	20	27	23	32	18	35	15	124	76
Number vomiting	5 (17%)	8 (40%)	3 (11%)	10(44%)	7(22%)	7(39%)	6 (17%)	1 (7%)	20(16%)	25(34%)
Mean age analysis (years)										
Vomiters	34.8		37.0		28.9		36.4		34.0	
Non vomiters	50.8		48.6		46.8		36.4		45.3	
Overall mean	46.9		45.6		42.1		36.4		42.5	

emetics in modifying the severity and time of vomiting, was assessed by grouping the vomiters in each drug group into multiple and single vomiters and calculating the mean time of occurrence of the first postoperative vomit.

Results

Effect of intra-operative antiemetics

The four groups of patients were comparable for age, sex, weight, duration of anaesthesia and the recovery ward time as shown in Table 1. Vomiting, sex and age analyses are shown in Table 2. The incidence of postoperative vomiting was 26% in each of the saline and droperidol groups, 28% in the metoclopramide group and 14% in the prochlorperazine group. The lower prochlorperazine incidence was not statistically significant. The overall vomiting incidence was 24% (47 out of 200).

Of those who vomited, droperidol reduced the number of patients having multiple vomits from 38% (saline) to 23% and prochlorperazine delayed the onset of vomiting from 4.9 hours (saline) to 7.6 hours. Females vomited more frequently than males overall in three of the four

Table 3. Postoperative oral fluids. Comparison of unrestricted and restricted postoperative oral fluid groups.

Postoperative oral fluid group	Sex and number			Age (years)		Weight (kg)		Duration of anaesthesia (minutes)		Recovery ward time (minutes)	
	M	F	Total	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Unrestricted	60	40	100	43.5	<1–85	48.7	4–79	72.0	25–190	41.3	5–180
Restricted	64	36	100	40.8	<1–82	47.6	7–77	67.2	20–135	36.7	15–95

Table 4. Postoperative oral fluids. The effect of restricting oral fluids on the incidence and time of vomiting.

	Unrestricted	Restricted	Total
Patient numbers	100	100	200
Number vomiting	20 (20%)	27 (27%)	47(24%)
Mean time of vomiting after surgery (hours)	4.7	5.6	5.2

Table 5. Postoperative oral fluids. The cause–effect relationship of first postoperative oral fluids on the incidence and time of vomiting.

	Unrestricted	Restricted	Total
Total vomiters	20	27	47
Number vomiting with first oral fluid	9 (45%)	13 (48%)	22 (47%)
Mean time of first fluid-associated vomiting(hours)	4.5	6.6	5.7

groups, with the exception of the prochlorperazine group in which more males vomited. Vomiting occurred in 34% (26 of 76) of females as compared to 16% (20 of 124) of males, a statistically significant difference ($p < 0.01$, Chi squared test).

Younger patients vomited more commonly in the drug groups and overall, again with the exception of the prochlorperazine group, with an overall mean age for vomiters of 34.0 years compared with 45.3 years for nonvomiters. This difference was statistically significant ($p < 0.05$). None of the patients vomited pre-operatively following morphine with atropine premedication and no extrapyramidal side effects of the antiemetic drugs were reported.

Eight patients received postoperative analgesia and subsequently vomited; the vomiting occurred between 3.75 and 11.5 hours after administration of the analgesia, with a median time of 5.5 hours.

Effect of postoperative oral fluid restriction

Comparison of the first 100 patients, in whom no definitive postoperative oral fluid restriction policy was enforced, with the second 100 patients, in whom oral fluids were restricted, revealed that these two patient groups were comparable for age, sex, weight, duration of anaesthesia and recovery ward time, as shown in Table 3. The incidence of vomiting in the 100 non oral fluid restricted patients was 20% and in the 100 oral fluid restricted patients, 27%; vomiting occurred at mean times of 4.7 and 5.6 hours postoperatively, respectively (Table 4). Neither of these differences was statistically significant.

Of those vomiting, the taking of first oral fluids after operation precipitated vomiting in nine (45%) of the 20 who vomited in the non oral restricted group and in 13 (48%) of the 27 in the fluid restricted group; the mean times of vomiting were 4.5 and 6.6 hours postoperatively, respectively (Table 5). These differences were also statistically insignificant.

Effect of surgical site

Statistics for the 200 patients after allocation into intra-ocular and non intra-ocular groups are shown in Table 6. Four patients had combined intra-ocular and non intra-ocular surgery, none of whom vomited or required analgesia postoperatively. The sex distribution of patients in the two main groups was similar, but those undergoing intra-ocular surgery were significantly older (Table 6). The mean duration of anaesthesia for intra-ocular procedures was 76 minutes compared with 55 minutes, and the mean recovery ward times were 42 minutes and 34 minutes for the intra-ocular and non intra-ocular groups, respectively.

The incidences of vomiting and postoperative analgesic administration related to surgical site are shown in Table 7. Twenty (16%) of the 123 intra-ocular patients vomited as compared with 27 (37%) of the non intra-ocular patients, a

Table 6. Surgical site: comparison of patients in non intra-ocular, intra-ocular and combined surgical gro

Surgical site	Sex and number			Age (years)		Weight (kg)		Duration of anaesthesia (minutes)		Recover ward tim (minute	
	M	F	Total	Mean	Range	Mean	Range	Mean	Range	Mean	Ra
Non intra-ocular	45	28	73	18.6	<1–72	33.1	4–76	55.3	20–185	33.9	5-
Intra-ocular	76	47	123	55.9	<1–83	57.5	4–94	75.8	20–190	41.9	15-
Combined	3	1	4	69.0	60–78	76.0	65–78	90.0	75–110	35.0	20-
Overall	124	76	200	42.5	<1–83	48.4	4–85	69.6	20–190	39.3	5-

Table 7. The relationship between surgical site and postoperative vomiting and analgesic requirements

		Non intra-ocular				
	Intra-ocular	Non-squint	Squint	Total	Combined	Tot
Total patient numbers	123	42	31	73	4	200
Number vomiting	20 (16%)	12 (29%)	15 (48%)	27 (37%)	0	47 (24
Number requiring analgesia	6 (5%)	7 (17%)	11 (35%)	18 (25%)	0	24 (12

statistically significant difference ($p < 0.01$, Chi squared test). In the non intra-ocular group, vomiting occurred in 15 (48%) of the 31 squint corrections, not quite significantly higher when compared to other non intra-ocular surgery, but highly significant when compared to intra-ocular surgery ($p < 0.001$).

Six (5%) of the intra-ocular patients required postoperative analgesia as compared to 18 (25%) of the non intra-ocular, which was a statistically significant difference ($p < 0.001$). Of the non intra-ocular patients, analgesia was required by 11 (35%) of the squints, a significantly higher requirement ($p < 0.05$) when compared with other non intra-ocular ($p < 0.05$) and intra-ocular procedures ($p < 0.001$).

Discussion

The undesirability of vomiting following eye surgery needs no elucidation and was illustrated during this study in two patients who, following postoperative vomiting, developed iris prolapses that required emergency surgery. The incidence of postsurgical emesis documented between 1955 and 1969 has been surveyed by Winning *et al.*,[16] who reported it as 30–68% patients. Purkis,[1] in a study of 1713 unspecified surgical patients, quoted an incidence of between 25 and 36%. The usefulness of various antiemetics (butyrophenones, phenothiazines, barbiturates, metoclopramide, diphenidol) in reducing these incidences following gynaecological,[17–23] orthopaedic[24] and mixed surgical procedures
has been extensively recorded.

Vomiting was reported by Borchert *et al*
occur with high frequency (66%) followin
surgery in children under 14 years of ag
wide differences exist in subsequently pub
reports, with incidences of 0–5% in males
reported by Nikki and Pohjola[2] and of 80-
being observed following squint correctio
children by Abramowitz *et al.*[3,29]

Droperidol has been shown to be an ef
antiemetic in ophthalmic surgery, but this
fit was not reproduced here and, surpris
none of the antiemetics had any significant
on postoperative vomiting. This may be d
the relatively small doses administered, a
because of the many patients at the extrem
age; this may also account for the absence o
observed extrapyramidal side effects of the
Droperidol, however, did slightly redu
number of patients who vomited more than
whilst prochlorperazine both delayed the o
vomiting and reduced its incidence, albeit
nificantly, from 26% (saline control) to
These reductions, if true, may be cli
important and have prompted continuing s
using prochlorperazine and droperidol.
clopramide had no beneficial antiemetic
This observation may support the view th
especially indicated for vomiting that
from gastrointestinal upset.[30]

No pre-operative vomiting followed mo
with atropine premedication. Of the 22 p

who received postoperative opioids, eight subsequently vomited. The latter is considered unlikely to be a cause–effect relationship as the vomiting occurred between 3.75 and 11.5 hours after administration of the analgesic.

The emetic precipitating effect of the first postoperative oral intake was reported by Burtles and Peckett,[31] mentioned by Parkhouse[32] and Bonica *et al.*[33] and cautioned against in ophthalmic anaesthesia by McGoldrick.[34] It is also taught to nursing staff, who usually dissuade postoperative patients from having oral fluids shortly after anaesthesia. The restriction of oral fluids in this study resulted in a slight but statistically insignificant increase in the incidence of vomiting from 20 to 27% and showed that the first oral intake precipitated vomiting to an equal extent both in those having early and in those having later access to fluids. This implies that delaying the time of the first postoperative oral intake simply delays the emetic precipitating effect of the first ingestate, but does not reduce it. Hence a denial of oral fluids postoperatively in ophthalmic surgical patients, in order to avoid vomiting, appears to be unjustified, although restriction is obviously advisable in patients still drowsy following anaesthesia.

The patients who had intra-ocular surgery were significantly older, had a lower incidence of postoperative vomiting and less requirement for postoperative analgesia. The low incidence of vomiting after intra-ocular surgery is fortunate as this is the group in which vomiting is most hazardous. Whilst the elderly, for undetermined reasons, are known to vomit less, their lesser analgesic usage may be due to stoicism and to the atraumatic nature of intra-ocular surgery. The higher incidence in the non intra-ocular group is explained by the relative youth of these patients, who have a greater tendency to postoperative emesis,[1,24] and by inclusion of the 31 squint corrections in this group, who had the highest incidence of vomiting.

The greater analgesic requirement in the non intra-ocular group, and especially in the squints (35.5%), reflects the more traumatic nature of non intra-ocular surgery and a possible lower degree of stoicism in younger patients. Severe surgical manipulation of the eye and the altered anatomical and physiological balances arising in the squint-corrected eye are presumed, via central reflexes, to account for the morbidity following squint surgery.

The overall vomiting incidence of 24% compares favourably with those reported in all surgical patients, although it is at variance with the high incidences popularly attributed to ophthalmic surgery.[2,3,7,8,28] Whilst not resolving the question of whether ophthalmic surgery is especially prone to cause vomiting, this categorisation delineates three groups of ophthalmic patients, namely an intra-ocular group with low predisposition, a non intra-ocular group with moderate predisposition and, within the non intra-ocular group, the squint group, with high predisposition. This high incidence following squint surgery appears responsible for all ophthalmic patients being described as especially prone to emesis.

Four conclusions may be drawn from this study. The antiemetics, droperidol, metoclopramide and prochlorperazine in the doses and manner described, are of no benefit in reducing the incidence or severity of postoperative vomiting when halothane has been breathed spontaneously. Restriction of oral fluids postoperatively does not reduce the incidence of vomiting, but merely delays it. Vomiting is precipitated by first oral intake in approximately 50% of those patients who do vomit irrespective of postoperative fasting. Ophthalmic patients may be allocated according to mean age and to the incidence of postoperative morbidity (vomiting and analgesic requirement) into three emetically predisposed groups, intra-ocular, non intra-ocular and, within the non intra-ocular, the squint group. Finally, this study finds that, with the exception of squint surgery, there is no justification for the opinion that postophthalmic surgery patients have a greater predisposition to, or incidence of, postoperative vomiting than that following other types of surgery.

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Economy in the use of muscle relaxants

Vecuronium after pancuronium

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Summary

Vecuronium is an intermediate duration, non-depolarising muscle relaxant. When used during prolonged procedures, it may be given by intermittent injection, by continuous infusion or by a single very large dose at the start of anaesthesia. It may also be used to maintain relaxation after the initial use of a long-acting agent, such as pancuronium. This study demonstrates that the effect of a dose of vecuronium used in this fashion is increased when interaction with pancuronium takes place. The clinical and theoretical implications of this observation are discussed.

Key words

Neuromuscular relaxants; pancuronium, vecuronium.

The neuromuscular transmission blocking drug vecuronium comes close to meeting the requirements for the ideal muscle relaxant proposed by Savarese and Kitz.[1] The short duration of effect and the rapidity of the reversal of blockade with or without an anticholinesterase make it suitable for short procedures.

Vecuronium has also been used for long procedures, when it is given either by intermittent injection, by continuous infusion[2] or by the injection of a single very large dose, given at the start of anaesthesia.[3] Although such methods of use may offer the advantage of the fast recovery of neuromuscular transmission seen after vecuronium, they require the use of large amounts of vecuronium and are therefore not economical. The initial use of a drug with a prolonged neuromuscular transmission blockade, followed by the use of vecuronium to maintain relaxation as the effects of the initial longer-acting drug wane, might combine the advantages of economy and rapid recovery.

The chemical structures of pancuronium and vecuronium are very similar. Pancuronium, which is slightly less potent than vecuronium, exhibits a similar relation between dose and response; the drugs are not synergistic and they may therefore have a similar site and mode of action.[4] In the present study, small incremental doses of vecuronium were given to maintain a clinically effective degree of neuromuscular transmission blockade after an initial dose of pancuronium. Observations were made with the aim of determining whether the advantage of rapid recovery normally seen after intermittent injection of vecuronium, was still present after the initial use of pancuronium.

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Method

Fourteen patients who were to undergo prolonged major abdominal surgery were studied. Two patients were given 2 mg lorazepam orally 2 hours before anaesthesia; the other patients were unpremedicated. Anaesthesia was induced using thiopentone and fentanyl 5–10 μg/kg given intravenously and maintained with nitrous oxide 70% in oxygen. Additional fentanyl was given when required.

Continuous monitoring of mean electroencephalographic (EEG) frequency and amplitude and frontalis electromyographic (EMG) activity was started before induction of anaesthesia, as were measurements of arterial blood pressure and pulse rate which were recorded automatically every minute.

Monitoring of neuromuscular transmission was started after induction of anaesthesia. The ulnar nerve was stimulated using surface electrodes at the wrist; a train of four supramaximal square-wave stimuli of 2 Hz was applied every 20 seconds. The integrated evoked compound action potentials of the adductor pollicis were recorded and displayed as bar graphs. After stable responses had been achieved, control values were recorded. The response to each initial stimulus of the train of four (T_1) was displayed digitally as a percentage of the initial control response.

Muscle relaxation was initially achieved by injecting pancuronium 55 μg/kg intravenously; the dose was established, using the same monitoring technique in a pilot study, to be approximately ED_{95}. The trachea was intubated and mechanical ventilation adjusted to maintain normocapnia; the end tidal $P\text{CO}_2$ was measured continuously. All monitoring and recording except for the continuous ECG, was with a Datex ABM.

In one patient the initial dose of pancuronium did not cause adequate muscle relaxation for the surgery envisaged. A supplementary dose of 0.5 mg was given intravenously 6 minutes after the initial dose. Muscle relaxation was subsequently maintained by intermittent intravenous injection of vecuronium 0.25 mg (nine patients) or 0.5 mg (five patients). Each dose was administered into a rapid infusion at the intravenous cannula as soon as a preselected value for T_1 was observed on the digital display as recovery of neuromuscular transmission occurred. Values of T_1 between 10 and 20% were used according to clinical requirements, but the increments were always given at the same level of response in each patient. After each increment the following measurements were recorded: the maximum (T_{max}) and minimum T_1 (T_{min}) and the times after injection when these values were first observed, and the time to the next increment of muscle relaxant. Recovery of neuromuscular transmission was allowed, towards the end of surgery, the time to the chosen T_1 level being noted but no increment given. Intravenous atropine and neostigmine were subsequently administered to hasten recovery of muscle function.

Results

The patient demographic data are recorded in Table 1. The effect, both mean and median, of the initial dose of pancuronium was a 93% block (T_1 = 7%, range 0–17%). The first increment of vecuronium was given 23.1 minutes (SEM 2.02) after the initial dose of pancuronium.

Table 1. Demographic data: means (SEM).

Group	*n*	Age, years	Weight, kg	Sex
0.25 mg	9	43.6 (3.2)	67.4 (5.5)	8F/1M
0.5 mg	5	47.4 (2)	59.8 (2.6)	4F/1M

Figure 1 illustrates the mean duration of effect of each increment of vecuronium. In every patient the duration of effect of the first dose of vecuronium was greater than that of any subsequent increment. The duration of effect of the first dose was significantly greater than that of any subsequent increment ($p < 0.01$, paired *t*-test). The mean duration of effect of the second injection was intermediate between those of the first and subsequent injections and significantly different from all other mean values ($p < 0.05$, paired *t*-test). The durations of effect of the third and subsequent increments were not different from one another. The relation between the duration of effect of each increment of vecuronium and the cumulative dose conforms to an exponential curve (correlation coefficient 0.9992).

The mean rate of use of vecuronium when given by incremental doses of 0.25 mg was 2.54 mg/hour; this reached 2.76 mg/hour when a steady state was achieved after the third dose. When increments of 0.5 mg were used, the over-

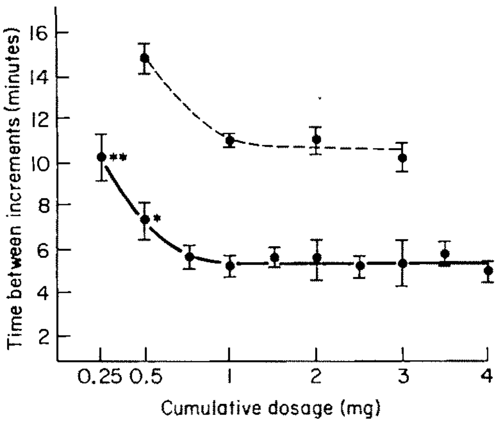

Fig. 1. Mean duration of effect T_{min} (SEM) of sequential increments of vecuronium 0.25 mg (● —— ●) or 0.5 mg (● – – – – ●) given at a constant level of neuromuscular blockade (80–90%) after pancuronium 55 μg/kg. ** Differs from subsequent values ($p < 0.01$, paired *t*-test); * differs from other values ($p < 0.05$, paired *t*-test).

all rate of use was 2.56 mg/hour, 2.79 mg/hour when steady state was reached.

The mean intensities of effect (decrease in T_1) of doses of similar magnitude given at different intervals (Table 2) or at different levels of neuromuscular block (T_1), were similar. The time to onset of maximum effect was also constant (mean 3.2 minutes, SEM 0.08).

Discussion

The results obtained in this study indicate that the duration of effect of a dose of vecuronium is increased when interaction with pancuronium takes place. The results may be explained on a theoretical basis. At the degrees of neuromuscular blockade at which vecuronium was first introduced, the muscle end-plate receptors would be expected to be still largely occupied by pancuronium; occupation of more receptors by vecuronium would cause an increase in blockade. The increase in transmission subsequently observed as receptors became free would be partially due to removal of vecuronium, but predominantly due to the removal of pancuronium. Assuming a fairly direct relation between plasma concentrations and effect, the apparent duration of effect of the first dose of vecuronium would be due less to the clearance of vecuronium, present only in very low concentrations and occupying relatively few of the receptors, than to the continuing slower clearance of pancuronium. However, as the pancuronium is cleared from the plasma with time and the proportion of receptors occupied by vecuronium increases with further dosage, the duration of effect of each increment becomes increasingly due to clearance of vecuronium.

The observations recorded here suggest that by the time the third increment of vecuronium 0.25 mg was given, the clinical effect of pancuronium was negligible, as the third and subsequent increments of vecuronium all had a similar duration of effect. The third increment of vecuronium 0.25 mg was given approximately 41 minutes after the administration of the ED_{95} dose of pancuronium, a duration of clinical effect of that drug that correlates with the experience of Noeldge *et al.*[2] who noted that 42 minutes were required for recovery of a pancuronium-induced block from 13 to 75% of transmission. However, it would appear that the initial dose of pancuronium was still exerting a residual effect more than 40 minutes after its administration and that the decline in this level of effect was slow. It would otherwise be difficult to explain how the very small total dose of 0.75 mg vecuronium given by that time was maintaining a neuromuscular transmission blockade in excess of 80–90%.

It could be anticipated that the intensity of effect of each increment of vecuronium would be similar in the same patient, irrespective of the time when it was given. Because the value of T_1 at which each increment was given was constant,

Table 2. Intensity of effect. Mean percentage decrease (SEM) in neuromuscular transmission (T_1) after each incremental dose of vecuronium.

	Increment							
Group	1	2	3	4	5	6	7	8
0.25 mg	4.18 (0.55)	3.9 (0.59)	4.8 (0.9)	3.5 (0.48)	4.2 (0.8)	2.9 (0.48)	3.1 (0.51)	2.6 (0.53)
n	9	9	9	9	9	9	8	7
0.5 mg	14.2 (0.97)	13.4 (2.2)	10.5 (2)	11 (5)				
n	5	5	4	2				

the degree of receptor occupation should be constant and the administration of a constant dose should then result in a constant increase in receptor occupation and intensity of effect. Whether the receptors were occupied by pancuronium or vecuronium would be irrelevant, assuming that the two drugs act in the same way at the same receptors.

The study demonstrated that the rates of use of vecuronium were the same when increments of either 0.25 or 0.5 mg were employed. Noeldge *et al.*[2] showed that the rates of use of vecuronium were similar whether patients were given intermittent doses of approximately 1 mg or an infusion in order to maintain a consistent level of neuromuscular blockade after a loading dose of vecuronium. However, in their study the rates of use were approximately 5 mg/hour, nearly twice the rates of use required in the present study. We can see no obvious explanation for this difference.

The ED_{95} for pancuronium suggested by the pilot study (i.e. 55 μg/kg) and supported by the present observations corresponds with the findings of Donlon *et al.*[5] and correlates closely with the results obtained by Ferres *et al.*,[4] although each of those groups measured mechanical twitch responses rather than the electromyographic responses used in the present study. All were less than the ED_{95} of 64 μg/kg indicated by Gramstad and Lilleaasen.[6]

The increased duration of effect of a dose of vecuronium when given after pancuronium was noted by Rashkovsky *et al.*;[7] however, their study reported only the effect of a single (cumulative) increment and did not reveal the time scale over which the interaction persists. This time scale is clinically important. If an anaesthetist wishes to follow pancuronium with vecuronium for reasons of economy during prolonged anaesthesia, and also wishes to take full advantage of the rapid clearance and short duration of effect of vecuronium, then it is necessary to introduce the vecuronium to maintain relaxation at a time during surgery which will allow the virtual elimination of the effect of pancuronium by clearance of that drug, i.e. 40 minutes to an hour before the estimated end of surgery. Despite this stricture, and ignoring the different cardiovascular effects of the two drugs, the sequential use of pancuronium and vecuronium has proved to be an attractive clinical routine.

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CASE REPORT

Anaesthesia and glucose-6-phosphate dehydrogenase deficiency

A case report and review of the literature

C. L. SMITH AND S. L. SNOWDON

Summary

Anaesthetic management of a patient with glucose-6-phosphate dehydrogenase deficiency is described. The pathogenesis and various complications relating to this common hereditary blood disorder are reviewed. Problems related to anaesthesia in the presence of glucose-6-phosphate dehydrogenase deficiency are discussed.

Key words

Genetic factors; glucose-6-phosphate dehydrogenase deficiency.

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common enzyme deficiency producing disease in humans,[1] but there are few reports[2,3] in the literature regarding anaesthesia in patients with this disorder. Haemolysis of red cells in G6PD-deficient individuals may occur after the use of a long list of oxidant drugs (Table 1) or during the course of various infections and metabolic disorders such as diabetic keto-acidosis.[1,4–7] It has been suggested that prilocaine[8,9] and possibly sodium nitroprusside[9] are contraindicated.

We describe the management of a case of known G6PD deficiency, followed by a discussion of the disorder with consideration of possible consequences in anaesthesia.

Case history

A 20-year-old female of African and Chinese heritage presented for plastic surgery to her face which was scarred by a house fire 11 years previously. G6PD deficiency had been diagnosed shortly after her birth, when she developed profound jaundice and anaemia with her haemoglobin falling to 6.7 g/dl by the 17th day. Her twin sister, who was also G6PD deficient, died in infancy probably of heart failure, the cause of her death being uncertain since it occurred at home. Subsequently a younger sister and brother were noted to be G6PD deficient. The family received counselling about forbidden medication and diet so as to avoid triggering a haemolytic reaction. Unfortunately the family has separated which makes elucidation of the G6PD status of each member impossible.

The patient had a past history of infectious hepatitis when 6 years old. No evidence of haemolysis was apparent during the illness. When 9 years of age, burns to her face and hands from the house fire necessitated admission to hospital. On the day after admission she had an

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Table 1. Drugs which may cause haemolysis in G6PD-deficient subjects.

- Antimalarials
  - Primaquine
  - Chloroquine
- Analgesics
  - Acetylsalicylic acid (only in very high dosage)
  - Acetophenetidin (phenacetin)
  - Acetanilid
- Sulphonamides
- Sulphones
- Nitrofurans
  - Nitrofurantoin
- Miscellaneous
  - Methylene blue; vitamin K (water-soluble analogues); nalidixic acid; naphthalene (moth balls); probenecid; phenylhydrazine; ascorbic acid in massive dose; nitrates
- Haemolytic in Mediterranean form of deficiency but not in A^- form
  - Chloramphenicol
  - Quinine
  - Quinidine

uneventful general anaesthetic for repair of flexor tendons of her forearm lacerated during escape from the fire. Over a 12-day period following admission her haemoglobin level decreased from 13.1 to 7.9 g/dl. A second general anaesthetic when 12 years old, for removal of a keloid nodule from behind her left ear, was uneventful. Anaesthetic agents used on these two occasions included thiopentone, morphine, tubocurarine, nitrous oxide and suxamethonium (on the second occasion). Her recent medical history had been uneventful except for an intermittently productive cough and haemoptysis, investigation of which showed her sputum to be clear of acid fast bacilli.

On the present admission for facial plastic surgery, pre-operative haemoglobin was 11.5 g/dl, reticulocytes 2% and a G6PD assay showed a level of 0.4 IU/g Hb (normal 3.3–5.7 IU/g Hb). Serum biochemistry was normal and chest X ray clear. Tests for the presence of haemoglobin S were negative. The G6PD assay confirmed her deficiency; the level was consistent with a homozygous state. The electrophoretic pattern of G6PD was typical of the African variant of the enzyme. Her only regular medication was the oral contraceptive pill. A list of contraindicated drugs was provided to the ward on her admission and it was particularly emphasised that she was not to receive any aspirin-containing preparations. Following advice from the haematologists two units of blood were cross matched to be ready in the event of significant postoperative haemolysis.

General anaesthesia was managed in a routine fashion: temazepam for premedication, induction with thiopentone, alcuronium for muscle relaxation followed by intubation of the trachea. Anaesthesia was maintained with nitrous oxide (500 ml/minute), oxygen (500 ml/minute) and isoflurane with monitoring of end tidal carbon dioxide and isoflurane levels. The anaesthetic gases were delivered through a carbon dioxide absorber system, with intermittent positive pressure provided by a Manley 'Servovent' to maintain end expiratory carbon dioxide partial pressures between 4 and 5 kPa. Continuous electrocardiographic monitoring and intermittent blood pressure measurements were performed.

It is usual practice for the plastic surgeon in our hospital to infiltrate the operative field with prilocaine 0.5% mixed with adrenaline 1:300 000. Because of the apparent contraindication[8,9] use of an alternative adrenaline-containing solution was suggested, though none was required.

Following an uneventful anaesthetic, the patient's haemoglobin level was measured daily for 4 days. On the 4th postoperative day she was discharged from hospital, as her haemoglobin had remained at the pre-operative level and electrolyte and liver function tests were normal.

Pathogenesis of G6PD deficiency

Glucose-6-phosphate dehydrogenase deficiency is inherited in a sex-linked fashion (see Fig. 1) and full expression of the defect occurs in males who carry the mutant gene on the single X chromosome and in homozygous females with the defect on both chromosomes. Female heterozygotes have two populations of red cells, deficient and normal, because during early development of the cell one X chromosome is inactivated in a random manner.[10] Consequently a condition of mosaicism of the red cells arises and the total level of G6PD in the blood of the heterozygous female may range from normal to that seen in the homozygous state.[1,4,5,7] More than 250 variants of G6PD deficiency are known[11] and new examples are

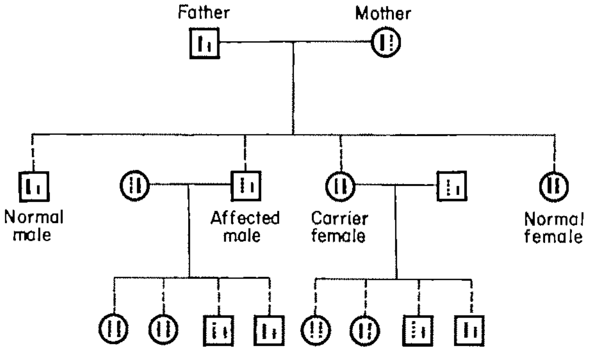

Fig. 1. X-linked transmission. ▮, Normal paternal X chromosome; ı, Y chromosome; ▯, normal maternal X chromosome; ⁞, defective X chromosome. If a male child inherits the defective chromosome, he will have the disease in the full-blown form. A female child on inheriting the defective chromosome may be clinically normal or have the disease to a variable degree, on expression of the two individual X chromosomes.

described in the literature at frequent intervals. Two forms of the G6PD enzyme are regarded as normal: the B form present in whites and most Africans, and the A (or A^+) form found in some Africans.[4] The most common of the G6PD deficiency variants is the A^- form seen in subjects of African descent. Among black Americans, the A^- gene frequency is approximately 11%.[1] The other relatively common variants of G6PD deficiency are the Mediterranean and Oriental forms, which although genetically distinct, behave in a similar manner.[12] G6PD-deficient individuals are essentially asymptomatic until stressed by drugs, infection or diabetic keto-acidosis. However, the Mediterranean and Oriental variants are generally associated with the more severe clinical problems and enzyme levels are usually lower than the African variant.[1,4–7] G6PD deficiency represents a serious public health problem in some parts of the world and the incidence in the Mediterranean basin ranges from 2.5—25%.[13,14] A recent study of African and Asian children in London showed a G6PD deficiency incidence of 9.3% in boys and 3.4% in girls.[15]

Young red cells with the A^- form of the enzyme initially have normal levels of G6PD and full enzyme activity is present, but as the cells mature the level decreases until no enzyme is found. If A^- individuals suffer an insult leading to haemolysis the resultant reticulocytosis provides an increased level of G6PD which limits the extent of the haemolytic process. In contrast, people with the Mediterranian and Oriental variants have decreased G6PD activity in both young and old red cells. Therefore, all red cells are susceptible to oxidative damage and a reticulocyte response does not reverse the haemolytic process while the triggering agent is still present. Haemolysis may consequently be more severe and life threatening.[4]

Clinically, anaemia and jaundice appear soon after the onset of haemolysis in G6PD deficiency. In severe cases, lumbar or abdominal pain occurs and the spleen is occasionally palpable.[5] Renal failure may result from haemolysis.[1,5,16] Severe haemolytic reactions often occur in Mediterranean variants of G6PD deficiency following ingestion of the bean *Vicia faba* (broad bean).[1,4,7] This condition, known as favism, particularly affects children[13] and may be fatal.[5,6] It is well recognised that G6PD-deficient cells provide some protective effect against the malaria parasite *Plasmodium falciparum*.[1,17] This protection is seen in heterozygous females who are genetic mosaics but not in homozygous males.[18] There is some evidence to show that the malaria parasite generates hydrogen peroxide (H_2O_2) within the red cell.[19] A G6PD-deficient cell is susceptible to the peroxidation, haemolyses and is rapidly removed from the circulation with resultant death of the parasite. However, it has been demonstrated that *Plasmodium falciparum* can undergo adaptive changes which enhance its ability to multiply when passed serially through G6PD-deficient red cells.[20] These workers have shown that *Plasmodium falciparum* can code a novel G6PD in deficient red cells which has a specific electrophoretic mobility. Therefore, homozygous

G6PD-deficient males are not protected but heterozygous females with both normal and G6PD-deficient cells are, as the chances of the malarial parasite infecting successive deficient red cells are much reduced.

Presentation of G6PD deficiency is not always typical and occasionally some rare types are associated with a chronic haemolytic anaemia.[7] Alternatively, non drug-related neonatal jaundice and anaemia may be the presenting feature and this, in fact, led to the diagnosis of the disorder in our patient.

Mechanism of haemolysis

Oxidant drugs cause increased generation of superoxide ion (O_2^-) and/or H_2O_2.[4,5] Both are potent oxidising species which can destroy the red cell. In addition, O_2^- can arise spontaneously from oxyhaemoglobin on the formation of methaemoglobin:[21,22]

$$\mathrm{Hb(Fe^{2+})\text{–}O_2} \rightarrow \mathrm{Hb(Fe^{3+})} + O_2^-$$

The cell is protected from O_2^- by the enzyme superoxide dismutase which converts O_2^- to H_2O_2.[23] The main defence against H_2O_2 in turn is provided by reduced glutathione (GSH) which, on enzymatic oxidation to the disulphide form (GSSG), results in the breakdown of H_2O_2 to water.[4] Reduced nicotinamide adenine phosphate (NADPH) is necessary to convert GSSG back to the reduced form so that the antioxidant capacity of the red cell is maintained (Fig. 2).

The sole source of NADPH in the red cell is the hexose monophosphate shunt, within which G6PD is a vital enzyme. The levels of GSH are not maintained without sufficient NADPH. Hence a red cell is susceptible to oxidant damage by O_2^- and H_2O_2 which results in haemoglobin denaturation and loss of red cell membrane integrity.[4]

Laboratory findings

The peripheral blood findings range from complete normality to those of acute haemolytic anaemia following an oxidant stress. Typically, drug-induced haemolysis begins 1–3 days after receipt of the offending agents.[5,7] The initial abnormality found is the presence of Heinz bodies in the red cells which, after appropriate staining, are visible under microscopy.[4,7] Anaemia soon follows, with hyperbilirubinaemia and haemoglobinuria depending on the extent of the haemolysis. As anaemia develops in the A^- form of G6PD deficiency, reticulocytosis occurs, arresting the decrease in haemoglobin and increasing G6PD levels. However, in the Mediterranean form the reticulocytosis may not lead to an increase in G6PD levels.

Glucose-6-phosphate dehydrogenase deficiency may be diagnosed by use of a quantitative assay. During an acute haemolytic episode a high reticulocyte count may produce a normal enzyme assay. Therefore, testing for suspected G6PD deficiency needs to be performed when the haemolytic episode is over.[4,7] Even with the Mediterranean type of deficiency, the red cell G6PD enzyme activity may increase during the haemolytic episode.[7] The various enzyme variants can be distinguished by electrophoresis.

Discussion

As observed with our patient, most individuals with G6PD deficiency are symptomless, unremarkable on physical examination and have a normal haematological picture. The clinical consequences of an insult that results in haemolysis, may be extremely variable. Anaemia may range from a very mild form which is apparent only after vigorous oxidant stress, to a form in which death results after massive haemolysis.[4] Various oxidant drugs, a wide variety of infections, and diabetic keto-acidosis may trigger haemolysis,[1,5–7] although recently the association between diabetic keto-acidosis and haemolysis in these patients has been queried.[24]

The avoidance of drugs which are known to precipitate haemolysis, is the mainstay of management of G6PD-deficient patients. If severe anaemia occurs then blood transfusion may be necessary. No reports were found in the literature of instances of acute haemolysis induced in these individuals by agents used for general anaesthesia. Nitrates may form part of the anaesthetist's armamentarium and isosorbide dinitrate has recently been shown to induce haemolysis in two patients.[25] These workers suggest that nitrate-induced haemolysis may be more commonly associated with G6PD deficiency than is suspected. The case of postanaesthetic prehepatic jaundice in a patient with G6PD deficiency described by Shapley and Wilson[2] did not receive any known oxidant

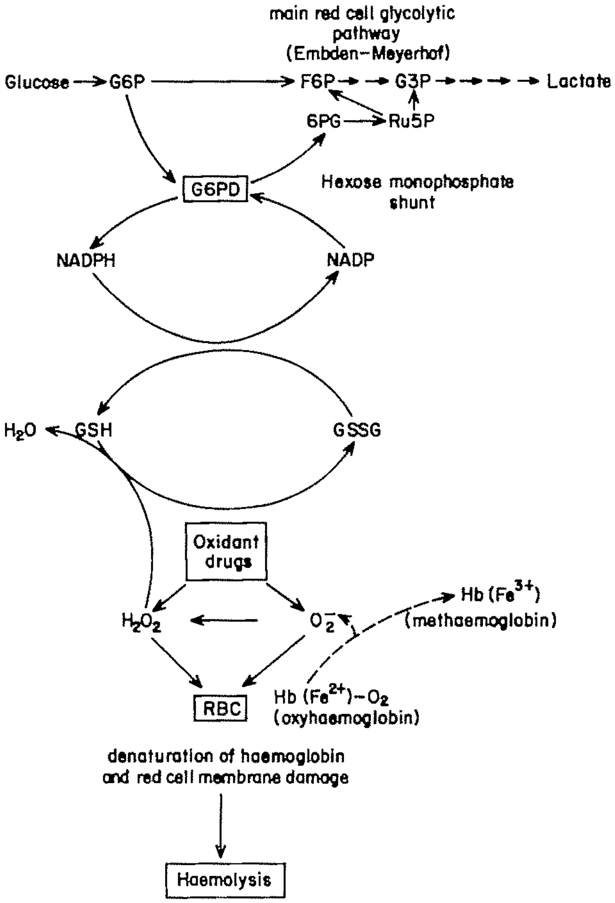
main red cell glycolytic pathway (Embden–Meyerhof)
Glucose
G6P
F6P
G3P
Lactate
6PG
Ru5P
G6PD
Hexose monophosphate shunt
NADPH
NADP
H_2O
GSH
GSSG
Oxidant drugs
H_2O_2
O_2^-
Hb (Fe^{3+}) (methaemoglobin)
RBC
Hb (Fe^{2+})–O_2 (oxyhaemoglobin)
denaturation of haemoglobin and red cell membrane damage
Haemolysis

Fig. 2. Illustration of oxidant protection in the red cell. The oxidants superoxide anion (O_2^-) and hydrogen peroxide (H_2O_2) may be formed from oxidant drug action or during methaemoglobin formation. Oxidant protection depends upon maintenance of normal levels of reduced glutathione by the enzyme nicotinamide adenine phosphate (NADPH), which in turn is dependent upon glucose-6-phosphate dehydrogenase (G6PD) function within the hexose monophosphate shunt. G6P, Glucose-6-phosphate; F6P, fructose-6-phosphate; G6PD, glucose-6-phosphate dehydrogenase; G3P, glyceraldehyde-3-phosphate; 6PG, 6-phosphogluconate; Ru5P, ribulose-5-phosphate; NADPH, nicotinamide adenine phosphate (reduced form); NADP, nicotinamide adenine phosphate (oxidised form); GSH, reduced glutathione; H_2O_2, hydrogen peroxide; GSSG, oxidised glutathione; O_2^-, superoxide anion.

drugs in the peri-operative period. Younker *et al.*[3] after reporting malignant hyperthermia in a patient with G6PD deficiency, postulated that the two conditions are linked via defective antioxidant enzyme systems, but confirmation of the diagnosis of malignant hyperthermia in the form of *in vitro* muscle tests was not given in the paper. Acetylsalicylate is often listed in the literature as being an oxidant drug which may lead to haemolysis in the presence of G6PD deficiency. Consequently our patient had in the past been urged to avoid aspirin, and the nursing staff during this recent admission were warned of the danger. When critically examined, however, aspirin has been found to be only slightly haemolytic even in large doses[1] and is said to be safe when administered in a dose of 50 mg/kg/day.[26]

Prilocaine and sodium nitroprusside have been described as contraindicated in the presence of G6PD deficiency[8,9] because it is claimed that methaemoglobin is not efficiently reduced in these patients.[8] Usage of prilocaine is increasing and this agent is now recommended as the local anaesthetic of choice for intravenous regional anaesthesia.[27,28] As methaemoglobinaemia is a possible result of using prilocaine, the contraindication described would seem important, since a significant proportion of people in the UK are of African, Mediterranean or Asian descent. However, the premise that G6PD-deficient patients are unable to reduce methaemoglobin from the ferric to the ferrous state, as suggested,[8] would appear to be untrue. Normally 3% of total haemoglobin is autoxidised to methaemoglobin daily,[29] but the serum methaemoglobin level is kept to less than 1% of total haemoglobin by a nicotinamide adenine dinucleotide (NADH) linked pathway.[30–32] NADH is a co-enzyme totally separate from NADPH, and its production is not affected by G6PD deficiency. While patients with a non-functioning NADH-reduction pathway will accumulate methaemoglobin,[33] a NADPH dehydrogenase-deficient patient did not have methaemoglobinaemia and his red cells were able to reduce the methaemoglobin induced by nitrite challenge.[34] Further evidence for the unimportance of NADPH in the reduction of methaemoglobin is provided by G6PD-deficient subjects themselves. Despite the inability to regenerate NADPH, they are able to reduce methaemoglobin and in fact have normal resting levels.[31,35]

Thus the evidence from these examples indicates that there may not be a sound theroretical reason why agents such as prilocaine and sodium nitroprusside in normal doses should not be used in patients with G6PD deficiency purely because they can cause methaemoglobinaemia. Nevertheless, the formation of methaemoglobin from haemoglobin results in the production of the oxidising chemicals O_2^- and H_2O_2 which may cause haemolysis in G6PD deficiency (Fig. 2). Therefore G6PD-deficient patients will be sensitive to a relative overdose of agents such as prilocaine and sodium nitroprusside with the resultant methaemoglobin formation. Hjelm and Holmdahl[36] found great individual variation in the methaemoglobin response to a given dose of prilocaine. Dosages as low as 5 mg/kg resulted in only slight elevations of serum methaemoglobin in most cases, but occasional subjects showed methaemoglobin values of 5% of total haemoglobin. Larger doses of prilocaine resulted in higher mean methaemoglobin levels and would certainly lead to a high risk of haemolysis in G6PD-deficient individuals because of their sensitivity to the oxidant byproducts of oxyhaemoglobin reduction. The usual prilocaine dosage for intravenous regional analgesia of the upper limb is 2–3 mg/kg body weight.[37] Provided this dose is not exceeded the increase in methaemoglobin production should be negligible.

Extra care is required with infants since methaemoglobinaemia is easily induced because of the low NADH dehydrogenase activity present.[38] The importance of this is illustrated by a newborn infant who developed methaemoglobinaemia after an 'acceptable' dose of prilocaine.[39] It is suggested that in the presence of G6PD deficiency, symptomatic methaemoglobinaemia in infancy should be treated by exchange transfusion, not with methylene blue.[38,39] Normally methaemoglobinameia responds promptly to treatment with methylene blue which, to be effective, requires conversion into its active form by NADPH.[40] In the presence of G6PD deficiency the blocked hexose monophosphate shunt does not regenerate NADPH and hence methylene blue is not effective in treating methaemoglobinaemia. In addition, methylene blue is an oxidant which has been incriminated as a trigger of haemolysis in a G6PD-deficient individual.[41] Thus, apart from being ineffective, the usual treatment for methaemoglobinaemia is contraindicated.

In conclusion, a review of the literature suggests that G6PD deficiency will not usually result in complications during or following anaesthesia, provided oxidant agents known to trigger haemolysis are avoided and patients with infection are observed carefully. Drugs such as prilocaine and sodium nitroprusside given in dosages which can accelerate methaemoglobin formation, will produce O_2^- and H_2O_2, potent oxidising species which cause haemolysis in individuals whose red cells are G6PD deficient. Evidence suggests that prilocaine in a dosage of 2–3 mg/kg would be unlikely to result in methaemoglobin formation but higher dosages may on some occasions. Therefore, whilst prilocaine may be suitable for intravenous regional analgesia of the upper arm in the presence of G6PD deficiency, if higher dosages are required for regional anaesthesia the the risk of haemolysis will be increased.

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Anaesthesia, 1987, Volume 42, pages 289–292

CASE REPORT

Respiratory obstruction following vocal cord injection

A complication on induction of anaesthesia

A. BODENHAM, R. LATIMER AND D. BETHUNE

Summary

Two patients who each had a paralysed left vocal cord are discussed. Both had poor coughing ability preoperatively. The affected cords had been injected with tetrafluoroethylene (Teflon) paste 3 days before operation and this had given immediate improvement in coughing and speech. During induction of anaesthesia, respiratory obstruction occurred that was related to the injected cord. This cause of obstruction has not been reported previously. The potential benefits of cord injection for voice, cough and laryngeal competence are reviewed.

Key words

Complications; respiratory obstruction.
Surgery; tracheal.

Case histories

Case 1

A 64-year-old man with widespread atheromatous vascular disease presented with a 2-year history of hoarseness, recurrent back pain and non-specific chest pains. He had a long history of smoking and was on metoprolol therapy for hypertension.

On admission, he was found to have disseminated vascular disease of the iliac vessels and aneurysms of the thoracic and abdominal aorta. Computerised axial tomography and aortograms demonstrated a saccular aneurysm of the arch of aorta. The paralysed vocal cord was attributed to compression of the left recurrent laryngeal nerve by the aneurysm (Fig. 1). He was booked for elective repair of his thoracic aneurysm and had his paralysed left vocal cord injected with tetrafluoroethylene (Teflon) 3 days before surgery. This procedure improved his voice and ability to cough.

The patient was premedicated with papaveretum 15 mg and hyoscine 0.3 mg intramuscularly and direct arterial monitoring was set up before anaesthesia. Induction followed with midazolam 2 mg, alfentanil 0.5 mg, nitrous oxide, oxygen and trichloroethylene while the patient breathed spontaneously. When the eyelash reflex was lost, 30 mg alcuronium was given. Complete obstruction of the airway followed and it was impossible to inflate the lungs manually with a mask and an oropharyngeal airway. At laryngoscopy the left cord was seen to be swollen and occupied more than half the glottic opening. A 39 French gauge Carlen's double lumen bronchial tube was passed with difficulty. Anaesthesia was continued with

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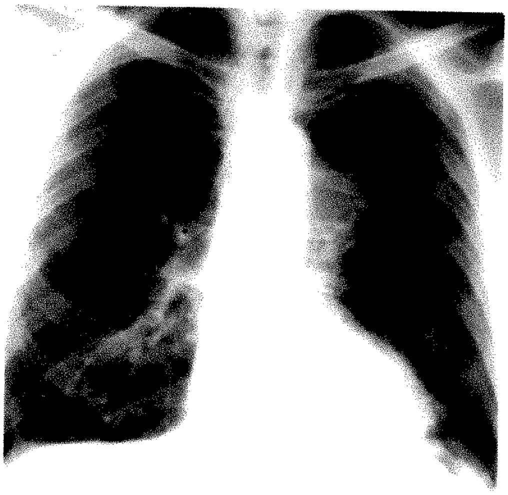

Fig. 1. Chest X ray showing thoracic aortic aneurysm.

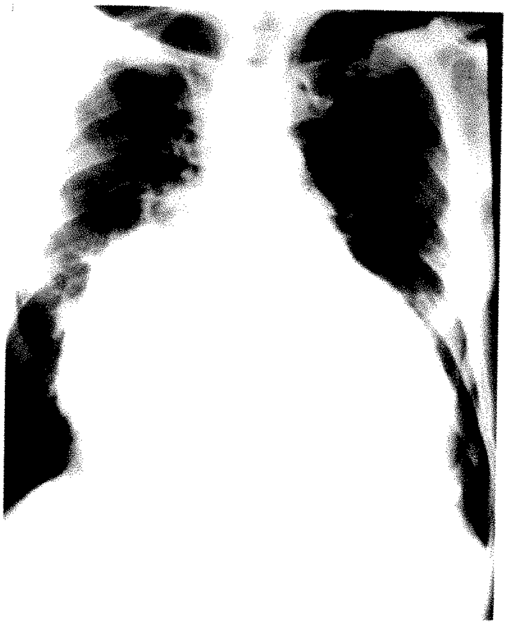

Fig. 2. Chest X ray showing gross cardiomegaly due to a giant left atrium and left ventricular hypertrophy.

nitrous oxide, oxygen and trichloroethylene and the lungs were easily inflated. An infusion of sodium nitroprusside was used to control the blood pressure.

A cannula was placed in the left femoral artery through a groin incision and a left thoracotomy was performed. After the left lung had been collapsed atriofemoral cardiopulmonary bypass was established. The aneurysm was resected and replaced with a 35-mm Dacron graft. The patient was weaned off bypass without difficulty and electively ventilated for 6 hours postoperatively.

Chest X ray after extubation showed a raised left hemidiaphragm consistent with a left phrenic nerve palsy. He was unable to clear chest secretions adequately, so a minitracheostomy (Portex Ltd) was inserted. This was used for 3 days to remove secretions and then withdrawn. Ten days postoperatively the patient was sent home in good health.

Case 2

A 68-year-old man presented with a 6-year history of increasing shortness of breath on exercise. Nine years earlier he had been noted to have a cardiac murmur. He had been hoarse for 12 months and could only speak in a whisper.

On examination he was dyspnoeic at rest, had an irregular pulse and signs of severe mitral regurgitation. Electrocardiogram confirmed atrial fibrillation and signs of left ventricular hypertrophy. His chest X ray showed gross cardiomegaly (Fig. 2). Echocardiogram confirmed severe mitral regurgitation and a giant left atrium. Catheter studies demonstrated normal coronary circulation and good left ventricular function. He was booked for mitral valve replacement. Three days pre-operatively his left vocal cord was injected with tetrafluoroethylene (Teflon) paste, which resulted in good restoration of his voice and coughing ability. The cord paralysis was attributed to compression of the left recurrent laryngeal nerve by the giant left atrium.

Papaveretum 20 mg and hyoscine 0.4 mg were given as premedication and direct arterial monitoring was set up under local anaesthesia. Induction was continued with midazolam 2 mg, nitrous oxide, oxygen and trichloroethylene; the patient breathed spontaneously. Alcuronium 30 mg was given when the eyelash reflex was lost. As in case 1, airway obstruction followed and it was impossible to inflate the lungs with a mask and an oropharyngeal airway. At laryngoscopy the left cord was seen to be grossly swollen and obscured most of the glottic opening. A 8.0 mm internal diameter cuffed tracheal tube (Portex) was passed without difficulty and the lungs could be easily ventilated.

Anaesthesia was maintained with nitrous oxide, oxygen and trichloroethylene. After median sternotomy the patient was established on cardiopulmonary bypass. The left atrium was drained of 1500 ml of blood and resected. The mitral valve showed myxomatous degeneration and was replaced with a 33 mm Bjork-Shiley prosthetic valve. The patient was taken off bypass and given an infusion of dobutamine and isoprenaline as inotropic support.

In the postoperative period the patient was ventilated for 48 hours until haemodynamically stable. His trachea was then extubated and he had good cough function and needed no further respiratory support. He was discharged for convalescence 15 days after operation.

Discussion

The recurrent laryngeal nerves may be damaged in the neck by malignant disease, cervical trauma or during thyroid surgery. The left nerve is damaged approximately twice as often as the right, because of its additional intrathoracic course. It may be damaged by malignant disease (usually bronchial or oesophageal) within the chest, an aortic aneurysm or giant left atrium secondary to mitral valve disease. The left nerve may also be damaged during surgery to a patent ductus arteriosus, because of their close anatomical proximity. A significant number of nerve palsies have no pathological or anatomical basis and are labelled idiopathic.[1]

Anaesthesia-related causes include tracheal intubation[2-4] and internal jugular vein catheterisation.[5] Cadaveric studies have shown that the anterior branch of the recurrent laryngeal nerve could be compressed between tracheal tube cuff and the thyroid lamina if the cuff is inflated within the larynx.[6]

A paralysed cord usually lies initially in the adducted position, at a slightly lower level than the non-paralysed cord due to altered muscular tone. With time, the cord atrophies and lies partially abducted.[7] The normal cord then cannot meet it in the midline and the patient develops a hoarse voice and loses the ability to cough effectively. With training, the normal cord may compensate by crossing the midline to meet the paralysed cord. However, this does not always occur, particularly in the elderly.

The shrunken cord can be enlarged by injection with tetrafluoroethylene (Teflon) paste, as first described in 1962.[8] Teflon fibres (7–100 μm) in a glycerine base are injected so that the leading edge of the vocal cord lies in the midline.[9] Surgical repair of damaged nerves is also practised in some centres. Anaesthetic techniques for laryngeal surgery have been reviewed and are not discussed here.[10]

Immediate airway obstruction in patients with an injected vocal cord has been reported in the anaesthetic literature, in a patient who had a cord injection under general anaesthesia.[11] In the two patients described in this report, the cord injections had been made under local anaesthesia 3 days before the events described, with an uneventful post injection period. Both of our cases of respiratory obstruction followed the onset of neuromuscular blockade with a nondepolarising relaxant. In neither case was any problem anticipated with the airway and our standard relaxant regimen of 30 mg of alcuronium for adult patients, provided rapid relaxation to allow intubation in an acceptable time. There was no clinical evidence of airway obstruction before neuromuscular blocking drugs were given. There was no obstruction after extubation, even when asleep. In both cases the injected cord was seen to occupy more than half the glottic opening. If it had been thought that there would be difficulty in the maintenance of the airway, relaxants would have been avoided for the initial laryngoscopy, which would have been performed under either local anaesthesia or inhalational anaesthesia.

Measurements of airflows through the larynx with a dry spirometer have been made before and after injection of a unilateral paralysed vocal cord.[12] Before injection, some airflow obstruction occurred in inspiration, due to the failure of the paralysed cord to abduct actively. By contrast, expiratory flows were not affected as the cord was passively abducted by the airflow. After injection, airflows were not significantly changed in expiration or inspiration, when compared to pre-injection values.

In both the cases reported here, the passage of the tracheal/bronchial tube was not significantly difficult. However, difficulties could be envisaged in the face of added distortion of the anatomy, as in cervical malignancy, where airway obstruction could cause a serious problem. It is known that Teflon injection initially causes swelling followed by local oedema and granuloma formation; later, the glycerine in the paste is absorbed and shrinkage occurs.[7] Rarely, an individual may have an idiosyncratic allergic-type response to the injected paste, that causes local oedema and obstruction.

It seems desirable, in the case of elective surgery, to allow the injected cord to shrink to its final size before further trauma is inflicted in the form of tracheal intubation. Double lumen tubes, in particular, may be traumatic on insertion. Guidelines as to the optimum time to leave between cord injection and elective surgery do not exist, but 2 weeks has been suggested as adequate. Alternatively, the paralysed cord may be injected postoperatively.

A paralysed vocal cord may be injected for palliative voice improvement in either benign or malignant disease. Additional benefits, such as improved cough and restoration of laryngeal competence, may also result.[13] Left recurrent nerve palsy due to malignant disease in the chest, signifies a poor surgical prognosis. However, in the case of a surgically correctable lesion, the patient may benefit from cord injection to improve coughing; this allows effective apposition of the cords, thus closing the airway. This produces the build-up in pressure within the airway necessary for effective coughing and the improved cough is of great value in lessening postoperative chest complications after thoracotomy.

The value of cord injection performed after oesophageal resection and anastomosis of the stomach to cervical oesophagus has also been reported.[14] Here there was delayed oesophageal emptying together with laryngeal incompetence due to a surgically damaged left recurrent laryngeal nerve. Cord injection, performed after operation, restored cough and laryngeal competence and so stopped passive aspiration.

In conclusion, vocal cord paralysis should be recognised pre-operatively. Cord injection may lessen postoperative chest complications by improved cough and laryngeal competence. In the presence of an injected cord, airway obstruction and difficulties in inflating the lungs should be anticipated. The optimum time interval between cord injection and surgery has not been determined but 2 weeks has been suggested as reasonable.

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Anaesthesia, 1987, Volume 42, pages 293–295

CASE REPORT

Spurious central venous pressure

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Summary

A patient is described in whom two different sites of cannulation of central veins produced markedly different pressures with potentially dangerous consequences. Falsely elevated pressures recorded from a catheter inserted via the subclavian vein were related to the patency in the arm of an arteriovenous shunt. The likely cause of this phenomenon is discussed.

Key words

Veins; venous pressure.

Prior to cannulation of a central vein, a decision must be made concerning the most suitable technique and route. The preferred approach usually depends upon the expected rate of success and the risk of producing injury to vital structures. It is generally considered satisfactory for the purpose of monitoring central venous pressure (CVP), that the tip of the catheter should lie within a great vein within the thorax.[1] The possibility of significant pressure gradients being found within these veins, apart from those necessary for the return of blood to the heart, does not appear to have been widely entertained. This case report indicates that sizeable gradients can occur under certain circumstances.

Case history

A 52-year-old man presented for renal transplantation and removal from the left upper arm of an infected Artegraft (bovine carotid artery employed here as a conduit for haemodialysis). His medical history was complex; in addition to chronic renal failure secondary to pyelonephritis, it included hypertension, diabetes mellitus, hyperlipidaemia, anterior myocardial infarction and subsequent coronary artery bypass grafting. Vascular access for dialysis had proven troublesome and his renal care physicians had commented on his tendency to become fluid overloaded despite haemodialysis.

Following an oral premedication of diazepam 15 mg, anaesthesia was induced with fentanyl 0.5 mg and thiopentone 100 mg given intravenously. Atracurium 30 mg provided muscular relaxation for tracheal intubation and subsequent intermittent positive pressure ventilation with oxygen and nitrous oxide.

A 16-gauge catheter was inserted without difficulty into the left subclavian vein via an infraclavicular approach. The readings of central venous pressure obtained by water manometry were in the range 25–30 cm H_2O above the mid-axillary line, prior to any significant fluid load. Even allowing for poor myocardial function and overhydration, these readings were considered excessively high and probably spurious, although respiratory pressure fluctuations were

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visible and blood could be freely aspirated from the catheter. A second 16-gauge catheter was inserted into the right internal jugular vein. This site had not been chosen initially because the patient had a very short and fat neck, but no difficulty was encountered in cannulation. This catheter appeared also to be in a satisfactory position, as gauged by the ready withdrawal of blood upon aspiration, and easily visible respiratory fluctuations.

However, whilst the pressure in the subclavian catheter was measured as 30 cm H_2O, that in the internal jugular catheter was only 15 cm H_2O, assessed in both cases by water manometry. This interesting situation persisted throughout renal transplantation. The readings obtained via the internal jugular catheter were considered more likely to represent right ventricular filling pressures and were monitored as such, whilst the subclavian catheter was used to administer a renal dose of dopamine (2 μg/kg/minute), the rate being regulated by a Sorenson 'Dial-A-Flo'. The Artegraft from the upper arm was clamped prior to its excision. The rate of infusion of dopamine was seen to accelerate dramatically immediately after clamping. This was promptly corrected, and the pressure now measured in this catheter was found to be within 1 cm H_2O of the pressure measured simultaneously in the internal jugular catheter.

After the operation, a chest X ray was obtained which demonstrated the positions of the two catheters (Fig. 1).

Discussion

The superior vena cava and brachiocephalic (innominate) veins are generally considered to be suitable sites for the tip of a catheter inserted for the measurement of CVP.[2] The subclavian vein has also been claimed to be a correct site,[3] although the presence of valves within the subclavian veins and the observation of pressure fluctuations within the subclavian veins consequent upon movement of the shoulders, imply that such positioning may be unsatisfactory for accurate pressure measurement.[4] It has also been noted that respiratory fluctuations are not an adequate indication of placement in a central vein, since these may occur when the catheter tip is extrathoracic, when falsely high pressures may also be obtained.[5]

However, the chest X ray supports the contention that both catheters were satisfactorily positioned to record central venous pressure in this patient. The catheter inserted via the infraclavicular approach lay along the line of the left subclavian and brachiocephalic veins, terminating in the midline behind the manubrium sterni, so that its tip should have been approximately at the midpoint of the brachiocephalic vein. The catheter inserted via the right internal

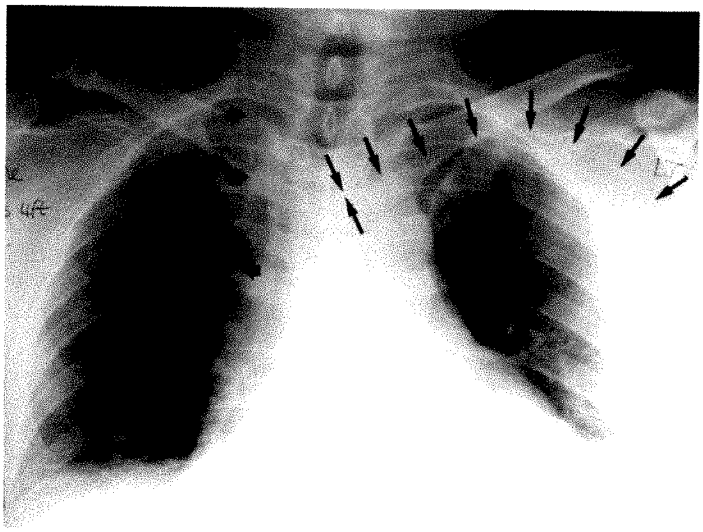

Fig. 1. Postoperative chest X ray. Arrows mark the positions of both catheters, the ends of which are indicated by opposing pairs of arrows.

jugular vein lay with its tip at the level of the third right costal cartilage, at the usual level of the junction of the superior vena cava and right atrium. The spuriously elevated pressures recorded from the subclavian catheter therefore must have depended upon an increased flow of blood within the brachiocephalic vein, and also a site for significant resistance to this flow between the two catheter tips.

The source of increased flow was obviously the Artegraft, which was between the brachial artery and an adjacent vein in the middle of the upper arm. Judging by its adequacy for haemodialysis, a minimum estimate of the flow in this would be 400 ml/minute. The cause or exact site of the venous resistance to this increased flow is less clear. Anatomical texts deny the existence of valves in the superior vena cava or brachiocephalic veins,[6,7] nor was there any evidence of pathology that might have caused external compression. The patient suffered from hyperlipidaemia, and venous mural plaques might conceivably have developed. It is also possible that the previous cardiac surgery might have resulted in venous scarring and distortion had caval cannulae been employed during cardiopulmonary bypass.

Whether or not there was any abnormality resulting in increased venous resistance, the increased venous return from an arteriovenous shunt in the arm resulted in the recording of spuriously increased pressures within a catheter that was inserted via the subclavian vein, and the tip of which lay in the brachiocephalic vein. This could have precipitated errors in fluid management, and also dangerous fluctuations in the rate of delivery of a powerfully vaso-active drug. It would appear prudent that careful consideration be given to the position of central venous catheters in patients with sizeable arteriovenous communications within the arm.

Acknowledgment

The author wishes to thank Mr C. Rudge, Consultant Urologist, Guy's Hospital, for permission to publish details of a patient under his care.

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Anaesthesia, 1987, Volume 42, pages 296–298

APPARATUS

Use of a low-power nerve stimulator during sciatic nerve block

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Summary

The use of a low-power nerve stimulator to aid nerve location during the performance of sciatic nerve block was compared with a conventional anatomically based technique. The success rate, both in terms of the production of an effective block and the duration of postoperative analgesia, was significantly higher in the stimulator groups than in the non-stimulator groups. The implications of the study for both clinical and teaching practice are discussed.

Key words

Anaesthetic techniques, regional; sciatic nerve block.

Sciatic block has earned a reputation amongst many anaesthetists as being both technically difficult and time-consuming to perform, with an often disappointing success rate.[1] This study was undertaken to investigate the clinical impression that a higher success rate was achieved when a low-power peripheral nerve stimulator[2] was used to aid nerve location during the performance of sciatic block, both in conscious and anaesthetised patients.

Patients and methods

Eighty patients undergoing surgery for femoral neck fractures were randomly allocated to one of four treatment groups. Formal consent was obtained and all patients were premedicated with lorazepam 2 mg orally, approximately 2 hours pre-operatively.

In group 1a, sciatic block was performed in conscious patients using a posterior approach based only on anatomical landmarks.[1] Paraesthesiae were actively sought in each case. In group 1b the same approach was used but, in addition, a low-power nerve stimulator[2] was attached to the blocking needle to aid location of the nerve (see below). Following sciatic blockade, general anaesthesia was induced with etomidate or thiopentone and maintained with halothane or enflurane, nitrous oxide and oxygen. No systemic analgesics were used during anaesthesia.

In groups 2a and 2b, general anaesthesia was induced as above prior to performance of the block. In group 2a patients, anatomical landmarks only were used to guide the blocking needle, while in group 2b, the nerve stimulator was used. The same posterior approach was used in both groups.

Technique for use of the nerve stimulator

In conscious patients (group 1b) the needle was introduced to a depth of approximately 1 cm. The active electrode of the stimulator was attached to the needle by means of a small croco-

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dile clip. The output power of the stimulator was increased gradually until the patient could report unequivocal perception of the stimulator pulses. The output power was fixed at this level and the needle advanced towards the nerve until pulse-synchronous paraesthesiae in the distribution of the sciatic nerve were elicited. Following a negative aspiration test, a 2-ml dose of the local anaesthetic agent was injected as recommended by Raj.[3] If the strength of the perceived pulses diminished within 30 seconds the full dose of local anaesthetic was injected over the following 30 seconds.

In unconscious patients (group 2b) the stimulator voltage was set to 7 volts (a value found to be necessary in a preliminary series to induce muscle twitching when the needle tip was in close proximity to the sciatic nerve) and the needle advanced towards the nerve until pulse-synchronous muscle twitching was observed in the calf muscles. A 2-ml dose was given, and if the amplitude of muscle twitching decreased within the following 30 seconds the full dose was injected.

The local anaesthetic agent used consisted of a mixture of equal parts of 1% prilocaine and 0.25% bupivacaine without adrenaline, at a dosage of 0.15 ml/kg body weight.

Assessment of the block was performed by an observer blind to the patient grouping. The efficacy of the block was tested in all patients on the basis of analgesia to pinprick in both the tibial and common peroneal nerve territories. Failure to block *both* territories was regarded as a failed block. Testing was performed on two occasions in all patients: first, approximately 30 minutes after consciousness was regained, provided that at least one hour had elapsed since induction of the block, and again at 8 hours postinduction.

Results

There were no significant differences between the four groups in terms of age, estimated weight, sex, duration of surgery or nature of surgery undertaken. The results of the initial postoperative testing are summarised in Table 1. The data were analysed using the Chi squared test with Yates' correction. The differences in success rate between the stimulator and non-stimulator groups are statistically significant (for groups 1a and 1b, $p < 0.05$; for groups 2a and 2b, $p < 0.005$).

Table 1. Patient allocation and number of sciatic blocks effective on initial testing. Groups 1a and 2a, anatomical approach only; groups 1b and 2b, nerve stimulator used.

Group	*n*	Effective block	
1a	18	8	$p < 0.05$
1b	21	19	
2a	23	6	$p < 0.005$
2b	18	16	

Table 2. Patient allocation and number of sciatic blocks still effective at the 8 hour testing. Groups 1a and 2a, anatomical approach only; groups 1b and 2b, nerve stimulator used.

Group	*n*	Effective block	
1a	18	6	$p < 0.005$
1b	21	18	
2a	23	5	$p < 0.005$
2b	18	14	

The results for the 8 hour postblock testing are summarised in Table 2. Again the differences between the stimulator and non-stimulator groups are statistically significant ($p < 0.005$ in both cases).

Discussion

These results suggest that the use of a low-power peripheral nerve stimulator[2] significantly improves both the success rate in establishing sciatic nerve block, as judged by the initial post-operative testing, and the duration of post-operative analgesia, as judged by the 8 hour postinduction testing, even in the conscious patient (groups 1a and 1b). Both of these effects presumably resulted from a more accurate de-position of the local anaesthetic agent in relation to the sciatic nerve.

When sciatic nerve block is performed in the conscious patient it is generally taught that paraesthesiae should be sought.[4,5] It is worthy of note that in group 1a, despite an often protracted search for paraesthesiae, these were elicited in only seven of the 18 patients (39%), and that only four of these appeared to result in a successful block as judged by the initial post-operative testing, with only three still effective at 8 hours. In contrast, in group 1b, pulse-synchronous paraesthesiae were obtained relatively easily in all 21 patients with a significantly higher incidence of successful block (19 of 21 patients, 90%). This figure is similar to that reported by Raj *et al.*[1] in their series in which almost twice the volume of local anaesthetic agent was used.

In group 2b, 16 of 18 patients (89%) showed an effective sciatic block at first testing. The ability to perform sciatic block in the anaesthetised patient may increase the clinical applications of the block, particularly for post-operative pain relief. In paediatric practice, for example, it is unlikely that an effective sciatic block could be performed on a conscious patient. Similarly, many elderly, confused or uncooperative patients might be regarded as unsuitable candidates for this type of anaesthesia. The ability to perform the block by using pulse-synchronous muscle twitching in a heavily sedated or anaesthetised patient may overcome many of these problems. General anaesthesia is also preferable when siting a catheter within the neurovascular compartment for continuous sciatic block.[6]

Teaching of local anaesthetic techniques

It was observed during the course of this study that trainees in anaesthesia were noticeably more willing to attempt sciatic block in the unconscious rather than the conscious patient. This appeared to be due to the lack of pressure on the trainee that otherwise occurred with an awake, apprehensive patient.

It would seem reasonable, therefore, to encourage the use of a peripheral nerve stimulator with unconscious patients when teaching trainees to perform sciatic nerve block in order that they may acquire a high degree of practical ability and confidence in a relaxed environment before attempting the block in a conscious subject.

Acknowledgments

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Anaesthesia, 1987, Volume 42, pages 299–304

APPARATUS

Drawover anaesthetic systems

Factors determining the inspired oxygen concentration

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Summary

The factors that determine the inspired oxygen concentration delivered by drawover anaesthetic systems were studied in the laboratory and in anaesthetised patients. The inspired oxygen concentration is dependent upon oxygen flow, reservoir volume and total ventilation; respiratory rate and inspiration : expiration ratio have a small influence. A reservoir of at least of 1 m corrugated tube (internal volume 415 ml) makes optimal use of a 4 litres/minute oxygen flow. Delivery of a safe inspired oxygen concentration can be ensured with economical oxygen flows.

Key words

Anaesthesia; inhalation.
Equipment; drawover.

Drawover anaesthetic systems use ambient air as the principal component of carrier gas. The air is supplemented by oxygen added to a T-piece with an open-ended reservoir tube on the inlet side of a low-resistance vaporizer (Fig. 1). The patient connexion is a dual-purpose non-rebreathing valve. This design is simple, safe and economical of compressed gas supplies. Drawover systems are therefore portable and suitable for independent use in remote locations. A similar design for oxygen enrichment is used at the air inlet of self-inflating resuscitators and of ventilators such as the Penlon Oxford.

Akinyemi and Adelaja[1] measured the arterial oxygen tension in five patients who breathed 5% halothane and air supplemented with 1 litre/minute oxygen from a drawover system, and found a mean of 29.3 kPa (SD 5.6). The FIO_2 was not measured. Because of the internal resistance of drawover anaesthetic systems, a small leak around a facemask leads to air dilution of the inspired mixture since, unlike conventional systems, there is no flow of fresh gas at a small positive pressure. The experience of Borland *et al.*[2] may reflect this. Of 28 patients who breathed halothane and air with up to 4 litres/minute added oxygen, only six had an ear lobe oxygen saturation of 95% or more. However, it was not clear whether these patients were intubated and the reservoir size and FIO_2 delivered were not reported. These and other authors do not specify the desirable size of the reservoir, although their illustrations generally show it to be much less than 1 m in length.[3]

If none of the oxygen delivered to the system is wasted to atmosphere from the open end of the reservoir, there are no leaks around a facemask and the total ventilation is known, then the FIO_2 can be predicted since the inspired mixture is composed of oxygen (known flow)

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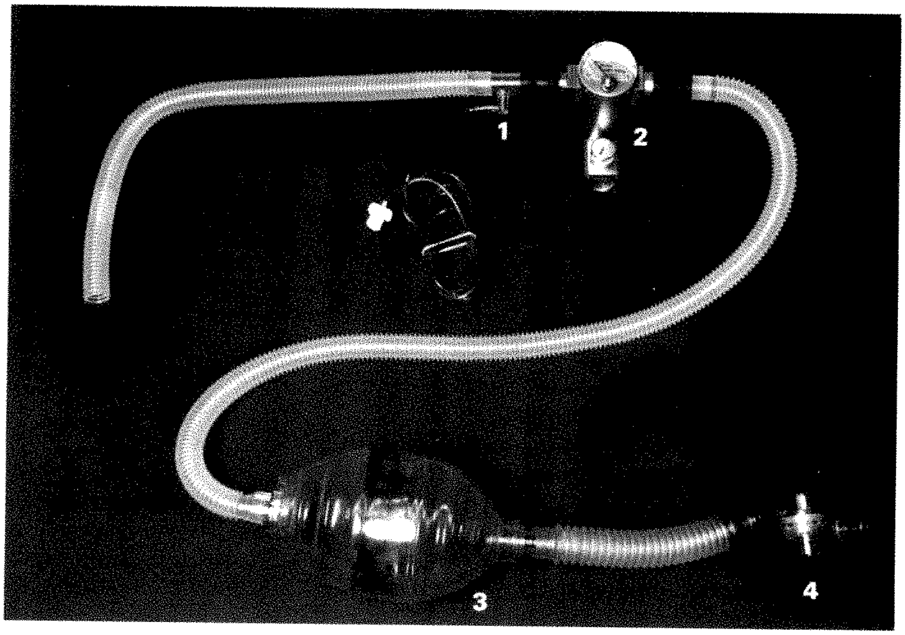

Fig. 1. Components of a drawover anaesthetic system. Oxygen is added to a T-piece and open-ended reservoir (1) on the inlet side of a low-resistance vaporizer (2). Gas mixtures pass to the patient through a self-inflating bag (3) and non-rebreathing valve (4).

and air (minute volume − oxygen flow).[4] Thus:

$$F\text{IO}_2 = [(\text{oxygen flow}) + 0.21(\dot{V} - \text{oxygen flow})]/\dot{V}$$

where $\dot{V}$ = total ventilation and 0.21 = $F\text{O}_2$ of air.

This study describes the factors that influence the $F\text{IO}_2$ delivered by drawover anaesthetic systems and the conditions necessary for the equation above to predict $F\text{IO}_2$ correctly. In the laboratory the effects of altering oxygen flow, reservoir volume, minute volume, inspiration:expiration ratio (I:E) and respiratory rate (f) were studied. In the patient study the effects of altering oxygen flow and reservoir volume were examined.

The oxygen flows selected (1 and 4 litres/minute) were the same as those delivered by the Houtonox valve, a component of the Triservice apparatus (Penlon, UK).[5] This device is a robust pressure-regulating valve in series with a choice of two resistances and is more rugged than a glass flowmeter.

Method

Laboratory study

In the laboratory study, the patient port of a Laerdal non-rebreathing valve was connected to the neck of a compliant bag suspended in a 21-litre glass bottle. Spontaneous respiration was simulated by generating calibrated gas flows into and out of the bottle, thus filling and emptying the bag. The gas flows were switched by a timing program on a BBC microcomputer that operated two solenoid valves. This arrangement permitted frequency, total ventilation and I:E ratio to be varied. Oxygen was delivered to the T-piece of a Triservice apparatus by each of the four possible combinations of two flows (1 and 4 litres/minute) with either of two lengths of reservoir, of internal volume 104 ml (25 cm) and 415 ml (100 cm). Three minute volumes were studied (4, 6 and 8 litres/minute) at two frequencies (10 and 20 breaths/minute) and two I:E ratios (1:2 and 1:1).

Total ventilation was measured with a bell spirometer. Oxygen was delivered from a Rotameter calibrated against an RT200 calibration analyser (Timeter Instrument Corp.). The oxygen concentration was measured at the expiratory side of the non-rebreathing valve with a calibrated Roche oxygen electrode and recorded 10 minutes after any change in the pattern of ventilation or oxygen supplementation.

Patient study

Twenty fit patients who presented for elective orthopaedic surgery for whom controlled ventilation was not required, were anaesthetised in a similar manner. They received 10 mg diazepam by mouth one hour pre-operatively (5 mg if less than 50 kg). Induction of anaesthesia was with intravenous thiopentone 5 mg/kg, suxamethonium 1 mg/kg and lignocaine 1 mg/kg. After intubation of the trachea, the patient's lungs were manually ventilated with air/oxygen, trichloroethylene (1%) and enflurane (3%), until the pupils were small. They then breathed spontaneously a mixture of trichloroethylene (1%) and enflurane (2%). A Triservice apparatus was used to deliver the anaesthetic mixtures.

Starting at the time of incision, oxygen was delivered for 10 minutes each by the same four combinations of 4 litres/minute or 1 litre/minute oxygen flow with a large (415 ml) or small (104 ml) reservoir, in random order, unless surgery was completed before all four had been used. F_{IO_2} was measured with a calibrated Roche oxygen electrode at the inspiratory side of the nonrebreathing valve, and end tidal carbon dioxide by a Datex infrared analyser sampling from the tracheal tube, as an indicator of any respiratory depression. Both were recorded at the end of each 10-minute period.

Results

Laboratory study

The results of the laboratory study are presented in Figs 2 and 3. Figure 2 shows the results for 4 litres/minute oxygen at a respiratory frequency of 10 breaths/minute. The effects of varying total ventilation, reservoir size and I:E are presented. The large reservoir delivers a higher F_{IO_2} than

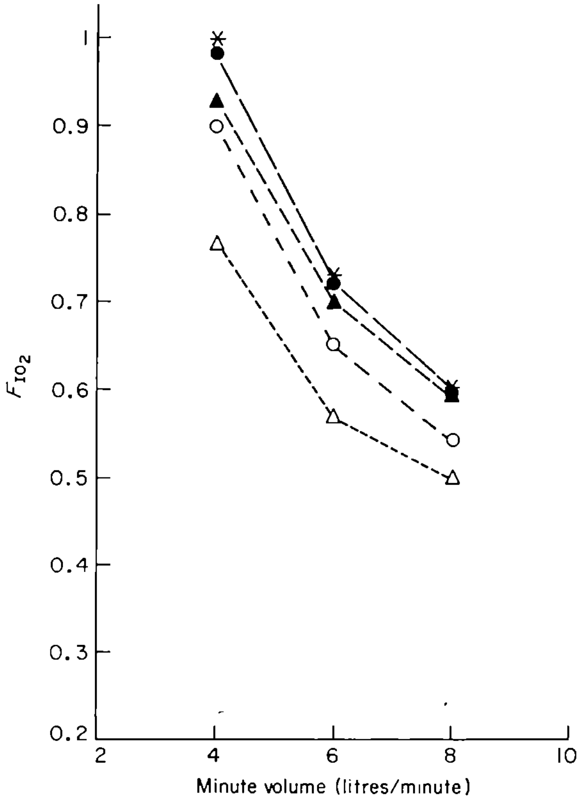

Fig. 2. Laboratory study, 4 litres/minute oxygen, f = 10 breaths/minute. The combination of a large reservoir and I:E of 1:1 delivers an F_{IO_2} close to that predicted by assuming no oxygen is wasted. Use of a small reservoir or I:E of 1:2 reduces the F_{IO_2}. *Small reservoir*: △, I:E 1:2; ▲, I:E 1:1. *Large reservoir*: ○, I:E 1:2; ●, I:E 1:1. *, Predicted F_{IO_2}.

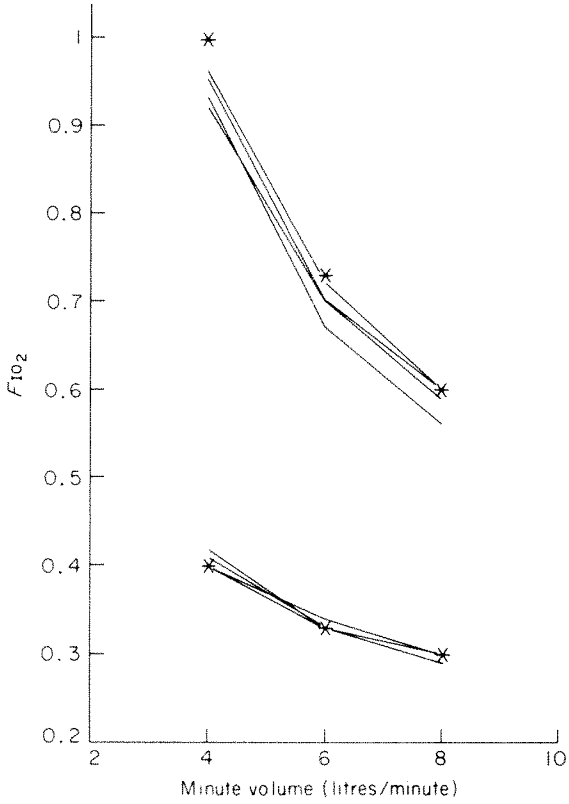

Fig. 3. Laboratory study. *Upper curves:* at 20 breaths/minute, when 4 litres/minute oxygen are added, changing I:E or reservoir size has little effect on F_{IO_2}. *Lower curves:* when only 1 litre/minute oxygen is added, frequency, I:E and reservoir size do not affect F_{IO_2}. Under these conditions F_{IO_2} is as predicted assuming no oxygen is wasted (*).

Table 1. The fractional inspired oxygen concentration predicted by the equation given in the text.

Minute volume (litres/minute)	Oxygen flow	
	1 litre/minute	4 litres/minute
4	0.41	1.0
6	0.34	0.74
8	0.31	0.61

the small and an I:E ratio of 1:1 delivers a higher F_{IO_2} than 1:2. The results can be compared with the F_{IO_2} predicted by the equation (Table 1). The F_{IO_2} calculated from the equation is closely approached by the combination of a large reservoir and an I:E ratio of 1:1. With a small reservoir and I:E of 1:2, the F_{IO_2} is only 77% of that predicted for a minute volume of 4 or 6 litres/minute and 83% of that predicted for 8 litres/minute.

In Fig. 3 the upper group of lines represents the results obtained with 4 litres/minute oxygen at a frequency of 20 breaths/minute. The differences in performance between the various combinations of reservoir size and I:E ratio are small compared to those seen at 10 breaths/minute. The lower group represents results with 1 litre/minute oxygen and all combinations of frequency, rate, reservoir size and I:E ratio. The F_{IO_2} in this group is not affected by these. Several of the 24 points and eight lines overlap. The F_{IO_2} is the same as that predicted by the equation.

Patient study

Twenty patients entered the study. Their mean age was 30 years (range 13–74) and mean weight was 65.8 kg (SD 13.4). Surgery was sometimes completed before all four combinations had been used, and 69 out of the maximum possible 80 readings were made. The results are summarised in Tables 2 and 3. The paired *t*-test was

Table 2. Results of patient study: all patients.

Oxygen flow (litres/minute)	Reservoir volume (ml)	Inspired oxygen concentration (%) Mean	Range	SD	*n*
1	104	41.4	36.0–51.0	4.98	20
1	415	42.0	29.0–50.5	5.96	18
4	104	82.9	67.0–96.0	8.84	16
4	415	90.3	61.0–96.0	8.88	15

Table 3. Results of patient study: paired results. Analysis of the paired results with 4 litres/minute supplemental oxygen shows that a significantly higher $F\text{IO}_2$ is delivered using the large reservoir. With 1 litre/minute oxygen the large and small reservoirs perform similarly.

Oxygen flow (litres/minute)	Reservoir volume (ml)	Inspired oxygen concentration (%) Mean	Difference	SE of difference	*n*	*t*	p
1	104	40.6	1.4	1.28	18	1.09	NS
1	415	42.0					
4	104	83.2	7.1	2.68	15	2.65	<0.02
4	415	90.3					

used to analyse these results, after unpaired data had been excluded.

At 1 litre/minute oxygen there was no difference between the $F\text{IO}_2$ delivered by the small or large reservoirs. The lowest $F\text{IO}_2$ recorded was 0.29. At 4 litres/minute oxygen the large reservoir delivered a mean $F\text{IO}_2$ of 0.9 and this is significantly different ($p < 0.02$) from the $F\text{IO}_2$ of 0.83 delivered by the small reservoir. The mean end tidal carbon dioxide was 5.6 kPa; the highest recorded was 6.9 kPa.

Discussion

In drawover apparatus when no gas is flowing through the vaporizer (expiration in a spontaneously breathing patient, but inspiration during manual ventilation), the oxygen added at the T-piece accumulates in the reservoir and displaces air from it. Eventually, oxygen will spill to atmosphere. Wasting of oxygen is facilitated by a low-volume reservoir and a long accumulation phase (low respiratory frequency, low I:E ratio in spontaneous ventilation). During the gas flow phase, oxygen and then air and oxygen together are drawn into the vaporizer and eventually to the patient. More air is drawn in at higher ventilatory volumes. The results of the present investigation show that total ventilation, oxygen flow and reservoir size are the major determinants of $F\text{IO}_2$. Respiratory rate and I:E ratio also influence it.

If, with up to 4 litres/minute oxygen, $F\text{IO}_2$ is to approach that predicted by the equation, a reservoir of internal volume 415 ml should be used. Intubated, anaesthetised patients who breathe spontaneously through drawover apparatus with an adequate reservoir are unlikely to have an $F\text{IO}_2$ of less than 0.3 with 1 litre/minute supplemental oxygen, or of less than 0.6 with 4 litres/minute oxygen. No significant respiratory depression was observed in the present study.

When a T-piece and reservoir are used to supplement air with oxygen at the gas inlet of a ventilator (e.g. the Penlon Oxford) or a resuscitator, the frequency and I:E ratio chosen may also influence the efficient use of oxygen.

Conclusion

A drawover anaesthetic system provides a satisfactory inspired oxygen concentration at economical flow rates. Air leaks around a mask should be avoided. A reservoir of internal volume 415 ml (1 m of standard anaesthetic hose) will make optimal use of a 4 litres/minute oxygen flow.

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Forum

Concurrent drug therapy in patients undergoing surgery

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Summary

In a retrospective study of 57 176 patients and a prospective study of 216 patients undergoing surgery, 24–32% of patients were receiving some concurrent medication. Between 10 and 16% were taking drugs for cardiovascular disease. Of these, only 71% had their normal medication prescribed before surgery and only 41% received their drugs on the day of surgery.

Key words

Drugs; pre-operative treatment.

Concurrent drug therapy is important in patients undergoing surgery because of possible interactions with anaesthetic drugs and because of the difficulty in maintaining normal drug therapy in the peri-operative period when patients may fast for long periods of time. We report a survey of patient compliance at this time.

Patients and methods

The anaesthetic records of 57 176 patients undergoing surgery in the Western Infirmary, Glasgow between April 1980 and November 1983 were reviewed previously.[1] On each form, the anaesthetist had recorded the presence and nature of any concurrent disease. In addition, coexistent drug therapy was noted. These data were filed on computer and retrieved subsequently.

In a prospective study of 216 consecutive patients undergoing general or orthopaedic surgery in two surgical units of the same hospital during April and May 1984, each patient was interviewed before and after surgery. Any concurrent disease and drug therapy were recorded. Regular medication was defined as more than 2 weeks' duration. Patient compliance with the regular medication on the day of surgery was determined from the nursing records. This was done without explaining the reasons to the nurses, in an attempt to avoid changing their normal practice.

Results

Of the 57 176 patients studied retrospectively, 13 703 (24%) were taking some medication regularly (Table 1). Just under half of these latter patients (5 999; 10% of the total) were taking drugs for some disease of the cardiovascular system. This group represents some two-thirds of the 8 618 patients undergoing surgery who had cardiovascular disease. In the prospective study, 32% of the patients were taking regular medication and half of these (16%) were taking drugs for disease of the cardiovascular system (Table 1). Of the

Table 1. Drug use in surgical patients.

	Retrospective survey	Prospective study
Patients studied	57 176 (100%)	216 (100%)
Patients on regular drug therapy	13 703 (24%)	70 (32%)
Patients with cardiovascular disease on regular therapy	5 999 (10%)	34 (16%)
Patients with cardiovascular disease	8 618 (15%)	—
Cardiovascular drugs prescribed before surgery	—	24 (71%)
Cardiovascular drugs given on day of surgery	—	14 (41%)

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patients receiving regular medication for cardiovascular disease, 71% had their medication prescribed and 41% received their medication on the day of surgery.

Discussion

Concurrent drug therapy is common in patients undergoing surgery. Some one in four patients might be expected to be taking some medication at the time of their operation. It is likely that drugs prescribed for cardiovascular disease are most relevant with respect to drug interactions with anaesthetic agents and our data suggest that one in ten patients are taking these drugs.

An unexpected but important result of our study is the frequency of acute withdrawal of concurrent medication before surgery. Both studies included patients undergoing elective and emergency surgery. The pattern of drug use was the same in both groups while the frequency of acute withdrawal of drugs was higher in the emergency patients (74 and 44% for emergency and elective patients, respectively). This may be hazardous for some drugs,[2] in particular β-adrenoreceptor antagonists[3] and calcium antagonists.[4] This aspect of drug administration requires further study to assess its role in the aetiology of tachycardia, hypertension and associated morbidity after surgery.

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Magill versus Mallinckrodt tracheal tubes. A comparative study of postoperative sore throat

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Summary

An attempt to assess the relative merits of Magill and Mallinckrodt tracheal tubes is described. One hundred patients scheduled for routine gynaecological operations were randomly allocated to one of two groups of 50; in one, Magill red rubber tubes were used and in the other, Mallinckrodt tubes. The incidence and severity of postoperative sore throat were assessed on the day after operation. Fifty percent of those intubated with Magill tubes suffered sore throats, compared with 28% with Mallinckrodt ($p < 0.05$). In both groups, sore throat was more frequent in younger patients undergoing short operations: smokers intubated with Magill tubes had significantly more sore throat than smokers in whom Mallinckrodt tubes were used.

Key words

Equipment; tracheal tubes.
Complication; postoperative sore throat.

Approximately seven thousand anaesthetics that require tracheal intubation are given in the Newcastle General Hospital each year; Magill red rubber tracheal tubes are used on about four thousand occasions, and PVC tubes in the remainder. This study was undertaken to assess the incidence and severity of postoperative sore throat with a view to formulating future equipment policy.

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Methods

One hundred female patients, all of ASA grades 1 or 2, scheduled for routine gynaecological operations, were randomly allocated to one of two groups of 50; in one group Magill tubes were used and in the other Mallinckrodt tubes, both types of 8 mm internal diameter. The two groups were broadly similar in age distribution (Fig. 1), type of operation, duration of intubation and cigarette consumption.

Intramuscular midazolam was used as premedication in most patients except in seven where oral temazepam was given—they had found this satisfactory in previous operations. Anaesthesia was induced with thiopentone and fentanyl. Tracheal intubation and muscle relaxation were achieved in short cases with vecuronium, and in long operations with alcuronium.

Maintenance of anaesthesia was with nitrous oxide and oxygen (2:1), enflurane 0.5–1% and supplements of fentanyl and relaxant as required. Atropine and neostigmine were used for reversal of residual neuromuscular blockade.

The tracheal tubes were lubricated with 5% lignocaine and hyaluronidase ointment; oropharyngeal airways were not used and anaesthetic gases were not humidified. The duration of intubation was noted. One patient was moderately difficult to intubate, but did not subsequently develop a sore throat. Three females were removed from the trial; one because a nasogastric tube was required during the operation, and two who later admitted to having had sore throats pre-operatively. All were replaced by similar cases.

The tube cuffs were inflated with air until a seal was just obtained. Pressure was recorded by a Portex cuff pressure gauge in the Mallinckrodt group and by a mercury sphygmomanometer in the Magill group. The sealing pressure was adjusted at 20-minute intervals.

All the anaesthetics were given by one of us (N.B.S.); the first 83 assessments were done by the other (P.L.A.), and the remaining 17 were performed by an independent anaesthetist. This change of assessor was necessitated by the move of P.L.A. to another hospital; there was, however, no statistical difference between the results of the two observers.

Patients were visited on the morning after operation by the assessor who enquired about how they felt generally, and then asked specifically about sore throat. Replies were graded as none, mild or severe. Bias was eliminated, as far as possible, because the interrogator did not know which type of tube had been used, and the anaesthetist was not aware of the former's findings until the end of the trial. Statistical analysis utilised either the Chi squared or Fisher's exact test.

Results

Sealing pressures in the Mallinckrodt tubes ranged from 0.5–4.4 kPa with a mean of 1.46 kPa, and in the Magill group the comparable figures were 5.4–40.8 and 20.8 kPa. It is not suggested that the latter represented the pressure of the cuff against the tracheal mucosa (C/T pressure), but the former figures probably do. Several ingenious methods of measuring C/T pressure have been described[1–5] but none is suitable for routine clinical use.

Fifty percent of patients intubated with Magill tubes developed sore throats compared with 28% in the Mal-

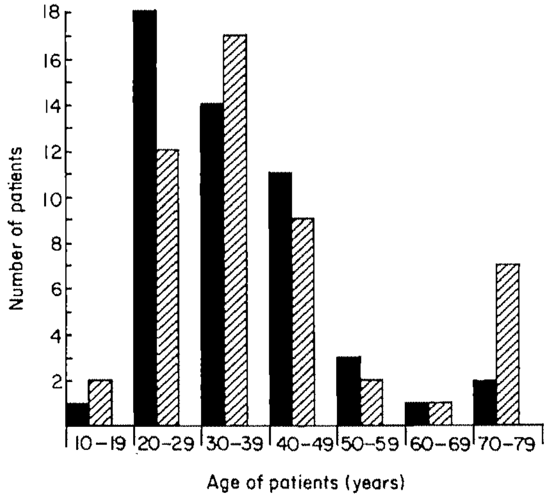

Fig. 1. Distribution of patients by age in two groups. ■, Magill; ■, Mallinckrodt.

Table 1. Incidence of postoperative sore throat in the two groups.

	Number of patients	Postoperative sore throat			
		None	Mild	Severe	Total
Magill	50	25 (50%)	19 (38%)	6† (12%)	25* (50%)
Mallinckrodt	50	36 (72%)	12 (24%)	2† (4%)	14* (28%)

Chi squared test. * Significant at $p < 0.05$. † Not significant.

Table 2. Incidence of postoperative sore throat related to age and duration of intubation (combined totals for two groups).

Age	Number of cases	Duration of intubation	Number of cases
16–39 years ($n = 64$)	35*	16–39 mins ($n = 46$)	27*
40–78 years ($n = 36$)	4*	40–119 mins ($n = 54$)	12*

Chi squared test. * Significant at $p < 0.01$.

Table 3. Incidence of postoperative sore throat in smokers and nonsmokers.

	Smokers				Nonsmokers			
	n	Mild	Severe	Total	*n*	Mild	Severe	Total
Magill	21	10	2	12* (57%)	29	9	4	13† (45%)
Mallinckrodt	19	4	0	4* (21%)	31	8	2	10† (32%)

Fisher's exact test. * Significant at $p < 0.02$. † Not significant.

linckrodt group, a significant difference ($p < 0.05$). Six patients in the Magill group had severe postoperative sore throat as against two in the Mallinckrodt, which is not statistically significant (Table 1).

Taking the combined figures for both types of tube, the incidence of sore throat was greater in the under-forty age group, and in those patients who were anaesthetised for less than 40 minutes, than in the older and more prolonged intubation series (Figs 2–4, Table 2). This rather surprising finding may be because most of the younger patients were undergoing laparoscopy which involved a steeper head-down tilt than that employed for hysterectomy or pelvic floor repair, with possibly more movement of the tracheal tube in the trachea.

In the Magill series 57% of the 21 cigarette smokers suffered from postoperative sore throat compared with 21% of the 19 smokers in the Mallinckrodt group (Table 3). (A smoker was defined as anyone who smoked at all, the average cigarette consumption being 15–20 per day.) This difference is significant ($p < 0.02$), and may be due to a sensitising effect that cigarette smoke may have on the mucosa for red rubber. In the nonsmoking group there was no significant difference in the incidence of sore throat between the two types of tube ($\chi^2 = 0.5$).

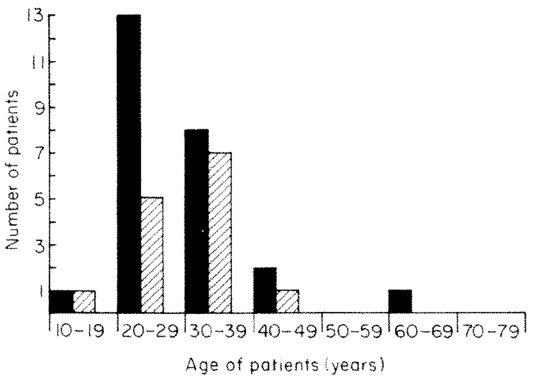

Fig. 2. Incidence of sore throat by age in two groups. ■, Magill; ▨, Mallinckrodt.

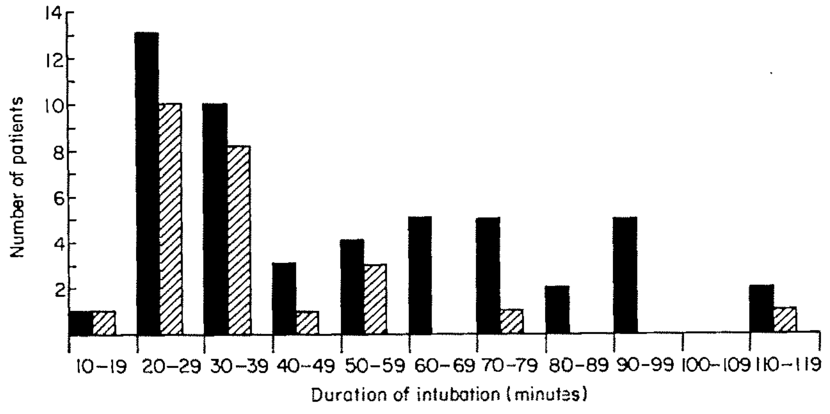

Fig. 3. Duration of intubation and incidence of sore throat with Magill tubes. ■, Patients intubated with Magill tubes; ■, patients developing sore throat.

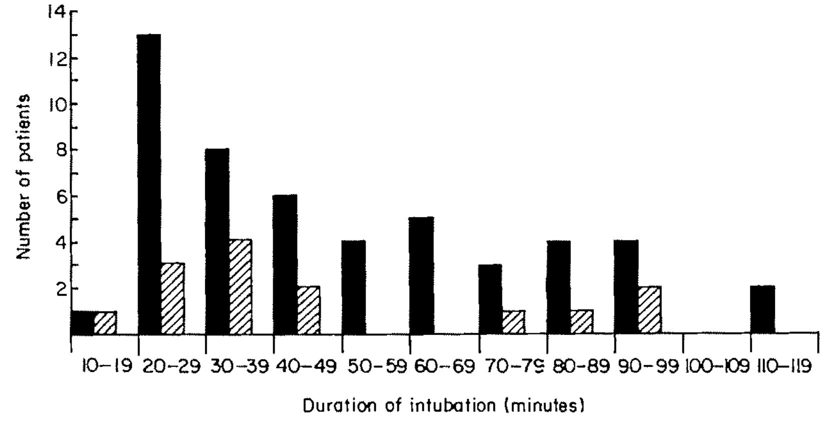

Fig. 4. Duration of intubation and incidence of sore throat with Mallinckrodt tubes. ■, Patients intubated with Mallinckrodt tubes; ■, patients developing sore throat.

Discussion

While an 81% incidence of postoperative sore throat has been reported,[6] 50% cannot be accepted complacently especially as Loeser and colleagues[7] achieved a rate of 10%. Intubation also poses a threat to the quality of the voice and it has been claimed that 3% of patients may sustain permanent damage.[8]

There are several factors which lead to sore throat,[9] but the two which have been studied most widely are tracheal tube cuff pressure and volume, and the area of contact between the cuff and the trachea. Tube cuffs are of two general designs: high-pressure, low-volume, low area of contact—of which the Magill red rubber is an example—and low-pressure, high-volume, high contact area—such as the Mallinckrodt tube.

In 1978 Loeser and co-workers[10] found that the lowest incidence of sore throat was obtained with low residual volume, low tracheal contact, high-pressur cuffs; high-volume cuffs produced more sore throat and these were of greater severity. This study wa performed on patients whose anaesthetics lasted 2– hours; the cuffs were filled with air, pressures an volumes were checked at the beginning and end of th operations and lignocaine ointment 5% was used as lubricant. A rider was added that the results migh not apply to longer periods of intubation.

Using a short (22 mm) low-volume, low-pressure low-contact cuff filled with inspired gas, the sam author[7] in 1980 reduced the rate of sore throat t 10% and stressed that morbidity was directly correlated with the length of the cuff, but not with duratio of intubation, age, type of operation or intracuf pressure. Other reports support the view that it is th area of tracheal contact which is important in preventing postoperative morbidity rather than cuff pressure, provided the latter is not excessive and is checked frequently.[11,12] Jensen and his team[12] make the point that the bulk of the high-volume cuff may damage the larynx during intubation and extubation. In our experience, most female larynxes will accommodate an 8.5-mm internal diameter red rubber tube but not a high-volume cuffed tube of similar diameter. In 1974 Mathias and Wedley[9] found that low-pressure cuffs produced less postoperative sore throat and this proposition has been supported by many observers since.[3,4,13-17] The circumference of a fully inflated high-volume cuff is greater than the internal circumference of the larynx and thus longitudinal folding results.[5,15] Mehta has pointed out the danger of incomplete seal at 'just seal' pressure because of this folding effect; his experiments were done on a Portex model and may not apply to clinical practice.[18,19]

It has been demonstrated repeatedly that an air-filled tracheal cuff absorbs nitrous oxide and oxygen with a consequent rise in internal pressure.[15,20-25] This can be avoided to some extent by inflating the cuff with anaesthetic gases or normal saline, but even these measures cannot be relied on completely because the proportions of inspired gases may alter during the anaesthetic and consequently cuff pressure may fall as well as rise. It is difficult to fill a cuff completely with saline, and absorption of anaesthetic gas into the residual air can lead to an increase in pressure.[26]

Loeser and colleagues[27] found that lubricating tubes with 5% lignocaine ointment gave the highest incidence of postoperative sore throat; best results were achieved with no lubrication, and intermediate rates were recorded when water-soluble jelly was used. The effect of lubrication was related to the area of tracheal contact and thus was greatest in high-volume cuffs.

Inflating the cuff with anaesthetic gases is not difficult, but is rather tedious and in our view unlikely to gain wide acceptance. It is simpler to fill the cuff with air, use a manometer, and correct pressure changes appropriately when they occur. Operations on the head and neck are often protracted and render the tracheal tube inaccessible; under these circumstances inflation of the cuff with saline may be the best answer.

We suggest that the incidence of postoperative sore throat can be minimised by using unlubricated, low-pressure, low area of contact cuffs inflated to 'just seal' pressure. Seegobin and Van Hasselt[15] have stressed that when such a tube is employed the cuff pressure must be kept as low as is compatible with effective sealing, and must not exceed capillary perfusion pressure (22–32 mmHg). This can be done by connecting the cuff line to a pressure gauge.

Acknowledgments

We are grateful to Drs S.P. Moffett and C.W. Thomson for their valuable help and advice, Dr C.A. Snodgrass and the nursing staff of the Gynaecological Unit for their cooperation, Mr A. McNay and Mr A. Beattie for statistical assistance, Messrs Mallinckrodt for supplying their tubes, to the University Graphics Section and to Mrs Shirley Tordoff who typed the manuscript.

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A matter of vigilance

In the continuing discussion on Dr Green's editorial (*Anaesthesia* 1986; **41:** 129–30) one is glad to see that Dr Eames has at last raised the question of the dependence placed upon the Operating Department Assistant (*Anaesthesia* 1986; **41:** 961). However, his comments did not go far enough.

In this country we train intensive care nurses to monitor unconscious patients, to infuse fluids at a variable rate to maintain a constant central venous pressure, to administer opiate and relaxant drugs as required, and to connect and disconnect patients from ventilators during periods of weaning. If we accept that this level of skill is commensurate with the actions of a person who is not medically trained, why do we hesitate so long in the use of non medically trained personnel to assist the anaesthetist in theatre? This step is contrary to the British traditions and would certainly require further training for the personnel concerned. However, it would allow anaesthetists to perform their bodily functions, which have been mentioned, without causing danger to the patient and might even allow the administration of a department of anaesthetics to continue normally!

Those of us who have worked in continental countries like Holland know how useful and reliable anaesthetic nurses can be. If such skilled help were trained in Britain, might this not end the charade by which anaesthetists claim to be permanently in theatre (while knowing that otherwise is the truth) and allow safer care of the unconscious patient and, it is to be hoped, fewer litigation claims?

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R.H. James

Having read and agreed with a recent editorial by Dr R.A. Green (*Anaesthesia* 1986; **41:** 129–30) in which it was stated that it is certainly not acceptable for a physician anaesthetist working on his own to leave an anaesthetised patient without supervision in the operating theatre during the course of an operation, I was pleased to receive my copy of the annual report of the Medical Protection Society. It was interesting to note that it had been considered 'impossible to justify' the absence of the anaesthetist from the table to answer a telephone passed to him in theatre, although he could see the monitoring apparatus;[1] absence from the theatre has rapidly been extended to absence from the table.

There is thus now a conflict between the vigilance required for patient safety and the time required to complete the detailed contemporaneous anaesthetic chart expected by prosecuting and defending lawyers. It is surely impossible to satisfy both demands at the moment. Until the use of reliable computers becomes widespread, my colleagues in the legal profession would do well to realise that the priority of the anaesthetist is patient care, not a perfect anaesthetic chart. In fact, a pristine anaesthetic chart could well indicate a lack of vigilance.

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J.M. McDowell

Reference

1. Medical Protection Society. *Annual Report and Accounts* 1986; **94:** 33.

A reply

Dr James has rightly pointed out the responsibility we give to trained intensive care nurses and compares their training with the average Operating Department Assistant (ODA). He also compares the training of our ODAs with the nurse anaesthetists with whom he has worked in Holland. If we were to train our ODAs up to these standards this would, I agree, relieve considerably the pressure on our anaesthetists. The facts are that, with very few exceptions, our ODAs and theatre nurses are not trained to these standards and therefore should not be left in charge of patients under anaesthesia. The average standard of anaesthesia in the United Kingdom is second to none and I certainly would not wish to swap it with a system involving nurse anaesthetists.

The writer sees no reason why anaesthetists should not be able to remain in theatre and thus provide safer care for their patients. The inference throughout this correspondence that anaesthetists have less control of their bodily functions than surgeons, who are unable to attend to these needs during the course of an operation, is, to me, puzzling.

In my original remarks on this matter I was, of course, referring to the dangers of the anaesthetist who habitually leaves his patients in the hands of untrained staff, for no very good reason, and to the consequences of doing so should something go wrong. There are, obviously, rare emergencies which would justify such an action.

Dr McDowell has emphasised a very important point which might well be digested by prosecuting lawyers involved in medical litigation. The writer has, on more than one occasion, tried to point out that a poor anaesthetic chart does not necessarily mean a low standard of anaesthesia. Indeed, as Dr McDowell has so rightly emphasised, a detailed contemporaneous anaesthetic chart may represent a degree of inattention to the patient's needs.

On the other hand, it must be appreciated that in the event of a claim being made for damage resulting from anaesthesia, the anaesthetic record may be the only evidence available on which to judge the progress of the anaesthetic, and possible liability for damage. As a result, the lack of good anaesthetic notes may, very often, lead to a judgement going against the anaesthetist. It is therefore important for the anaesthetist's protection that a comprehensive anaesthetic record is completed in every case. This record need not be contemporaneous, particularly in short cases, but should be completed as soon as possible after cessation of anaesthesia.

The routine use of an automated recorder is certainly recommended.

The Medical Protection Society,
50 Hallam Street,
London W1

R.A. GREEN

Cardiopulmonary resuscitation in district general hospitals

The function of a hospital cardiopulmonary resuscitation (CPR) service may be influenced by a variety of factors such as the composition of the resuscitation team, the availability of hospital-wide CPR treatment, the prevalence of non-resuscitation policies, methods of organisation of the CPR service and data collection. Investigations that demonstrate poor levels of expertise in CPR techniques among junior hospital doctors have led to recommendations for more training in this field.[1] A questionnaire, to record the pattern of practice with regard to the above factors and to assess the impact of recommendations on training, was sent to 18 district general hospitals of between 380 and 980 beds in England, Wales and Scotland. A consultant involved in the organisation of the service was contacted, the purpose of the questionnaire explained and confidentiality assured. All 18 agreed to participate but 16 questionnaires were returned.

The skills of several doctors are required for rapid and effective resuscitation, and the finding that CPR teams included more than one doctor in all hospitals is reassuring. However, the management of an obstructed airway, effective artificial ventilation and central venous cannulation are skills particularly relevant to anaesthetists. In three hospitals, junior anaesthetists were not included in the CPR team. It is clearly essential that all trainee anaesthetists receive both theoretical instruction and practical experience in CPR. Indeed, it is likely that the Faculty of Anaesthetists will be stressing the need for CPR training in their criteria for general professional training (J.S.M. Zorab, personal communication) and this should encourage their inclusion on CPR teams.

Specifically designated nurses or operating department orderlies were included in the CPR team in only 10 of the 16 hospitals. The presence of such staff usually ensures a high quality of assistance because of their familiarity with equipment and accumulated experience with the procedures. They may be drawn from sites such as ICU, CCU or the accident unit where resuscitation skills are frequently required. Their inclusion on CPR teams is recommended.

There may not be agreement on currently ideal methods of resuscitation, but inconsistencies in approach may have detrimental effects on results. The speed of CPR treatment is crucial for its success and there is no time to debate the merits of a particular clinical decision, or for misunderstandings between members of the team. These problems can be largely overcome by the adoption of a single policy on the management of CPR in a hospital. Such a policy should define the fundamental steps to be taken for the common causes of cardiopulmonary arrest and may take the form of a clear flowchart such as that published by the Resuscitation Council (UK). The benefits of implementing a policy include increased speed of treatment arising from familiarity with one sequence, and the maintenance of a consistent level of performance in a field where staff turnover is rapid. The policy may form the basis of a CPR teaching programme which will encourage its use. Only five of the sixteen hospitals had adopted such a policy and in all five this conformed to the guidelines of the Resuscitation Council (UK).

Published results of CPR have emphasised that a significant proportion of those who did not ultimately survive CPR should not have received this treatment at all.[2] Attempted resuscitation of patients for whom an unsuccessful outcome can be confidently predicted is inappropriate, since it achieves only unnecessary suffering for patients and relatives. It is unreasonable to expect a patient's prognosis to be evaluated and a decision not to resuscitate to be made during the resuscitative effort. The decision to withold CPR should be taken in advance by the consultant responsible for the patient's care and it should be clearly communicated. The finding that only one hospital of the sixteen had implemented a definite uniform policy on instructions not to resuscitate, indicates the difficulties in arriving at agreement on such a policy although there is little doubt that the need for these policies is great.[2,3]

An effective CPR service requires the coordination and cooperation of medical, nursing, technical and ancillary staff. It is therefore appropriate to involve some of these groups in the management of the service. In nine of the sixteen hospitals, organisation of the

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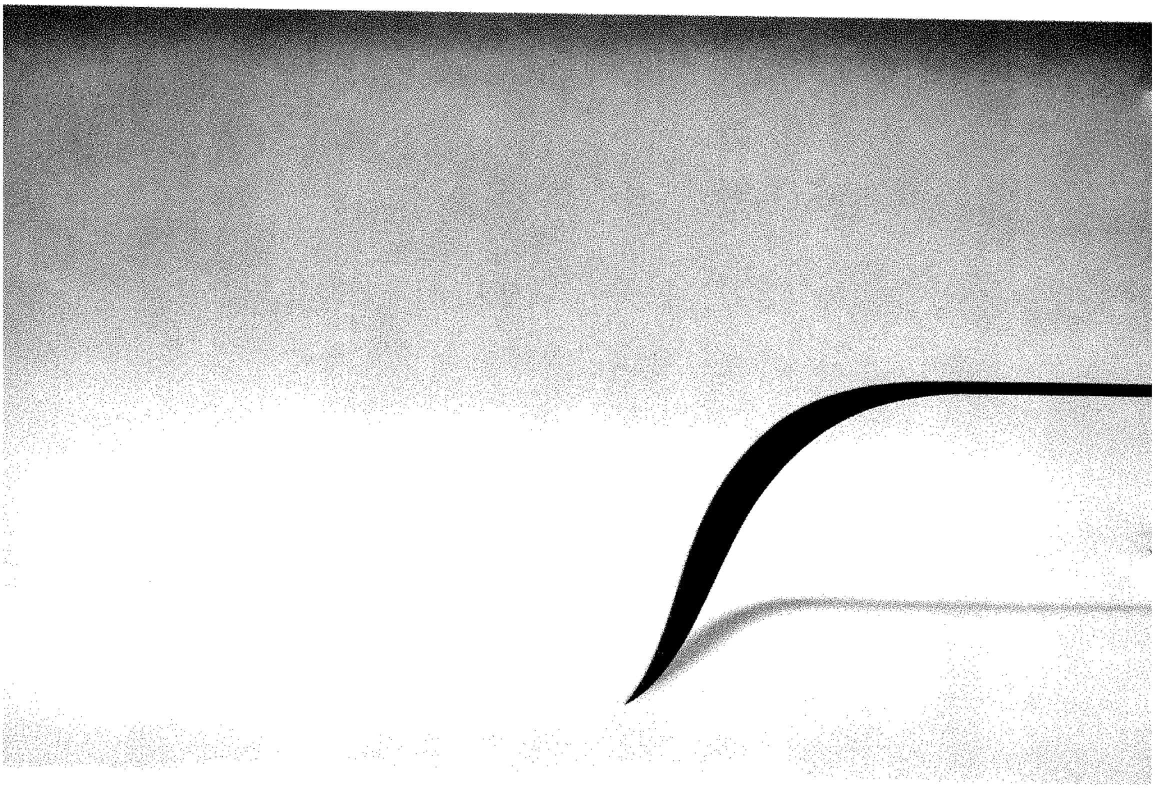

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service was by a multidisciplinary committee. In three hospitals the organisation was undertaken by one department and in four, by a single individual.

Assessment of the performance of a CPR service requires a dedicated system of data collection. This is an extremely labour-intensive task, so it is not surprising that a minority of hospitals was found to undertake it. The Resuscitation Council (UK) is currently reviewing the results from several centres in the UK and it is hoped that this will lead to a set of simple guidelines for assessment which will encourage better practice by CPR services.

Several studies have reported inadequate levels of expertise in the CPR techniques of junior hospital doctors[4] and recommendations for postgraduate teaching programmes have been made. The questionnaire did not enquire about the nature of the training programmes, but the finding that 13 of the 16 district general hospitals ran a training programme of some kind is encouraging.

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The severance of epidural catheters

We are grateful to Dr Hutchison for giving us a preview of his valuable work (*Anaesthesia* 1987; **42:** 182–5) on the strength of Portex epidural catheters. His extreme test prompts us to make two observations.

Catheter variables apart, the breaking loads of the catheter are dependent on the geometry of the back edge of the needle bevel, and its sharpness. In a matched set such as the Portex Minipack, this test is fair, since a virgin needle would be used on each occasion in clinical practice. However, with the still widespread use of re-usable needles, with occasional resharpening, we have found razor sharp edges which would shear a catheter much more easily than was achieved in Dr Hutchison's series. Variation has also been observed in this respect between virgin single-use needles from different manufacturers. We feel that some of the cases of catheter failure reported to us, have been as a result of the use of extremely sharp needles with Portex catheters.

The British Standard *Specification for sterile epidural catheters and their introducer needles for single use* (BS6196, 1982) states that introducer (Tuohy) needle packs should quote the legend 'Caution: Never pull back the catheter through the introducer needle'. This warning is precisely to draw the user's attention to the limitations of the equipment being used as a result of the bevel geometry, and to prevent the type of failure evaluated in this paper.

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A reply

Thank you for the opportunity to comment.

The role of the needle bevel in determining the breaking strain of the catheter is stressed several times in my paper. The use of a Portex catheter through any other sort of needle would almost certainly result in a different breaking strain from that reported for the corresponding Minipack set.

One therefore agrees with Mr Butchers' warning against applying the results of the tests described to any catheter/needle combination other than those supplied in the Minipack series.

It is noted in the paper's introduction that product information supplied with epidural sets mentions the danger of withdrawing the catheter through the needle. The intention of the investigation was to quantify that danger. If nothing else, inadvertent withdrawals will occur from time to time, and it is obviously desirable to have a high breaking strain, high stretch before breaking system to minimise the risk of catheter division in such an eventuality.

With the reservations discussed in the paper, Portex would appear to have provided such a system.

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Triazolam premedication

We have read with interest the paper on triazolam premedication by Dr Thomas *et al.* (*Anaesthesia* 1986; **41:** 692). In particular, the conclusion that triazolam 0.25 mg produced effective pre-operative anxiolysis is surprising in view of the results presented. There was no difference between the groups, but patients who received triazolam were judged to be less anxious than baseline while in the anaesthetic room. This appears to be the only evidence for the improvement in anxiety upon which the conclusion is based and, therefore, some further explanation is required.

We are told that one of the authors assessed the patients pre- and postoperatively, but it could be construed that the anaesthetist was the assessor in the anaesthetic room. A different assessor used at this time would serve to lessen the precision of the test and cast doubt upon the validity of the observation. The use of the linear analogue score (LAS) for anxiety has been established[1,2] but, as far as we know, this has been confined to use by the patient only in quantifying a subjective variable. Has the use of the LAS by an observer to measure subjective effects in the patient been evaluated?

It is regrettable that no account was taken of the patient's own assessment of anxiety. Forrest *et al.*[3] questioned the reliability of conclusions based solely on observer assessments of anxiolysis, in view of the disagreement between observers and patients in this measurement. In the study of Thomas *et al.*, no changes in perceived anxiety were noted by patients who received triazolam, and no reduction in anxiety was demonstrated using the multiple affect adjective check list (MAACL). This result accords with the patient assessments in the study by Pinnock *et al.*[4] and, therefore, conflicts with the overall conclusion in respect of the usefulness of triazolam.

Unfortunately, no data are given in Table 3 for the patient's assessment in the anaesthetic room, of sleepiness, but the observers rated all patients more sleepy than at baseline, with a mean increment in those who received triazolam of 350% as compared to 188% and 170% increments in the lorazepam and placebo groups, respectively. This would seem to indicate a trend in favour of a sedative and hypnotic effect of triazolam at this time.

Regarding recovery at 6 hours from undesirable side effects, no data are given for the linear analogue dizziness scores assessed by the patients 6 hours postoperatively, but we are told that all patients were less clear-headed (that is, more dizzy) than at baseline. If the influence of the increase in dizziness at 2 hours (850% for triazolam and 300% for the other groups) is apparent at 6 hours, then it would seem that triazolam could not be recommended for use in patients who are to be discharged at that time.

The authors state that: 'The need for pharmacological premedication is demonstrated by the anaesthetists who found a reduction in pre-operative anxiety in premedicated patients'. Does this indeed mean that premedication is given to satisfy the requirements of the anaesthetist in preference to those of the patient?!

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A reply

We should like to reassure Drs Pinnock and Fell that each patient was assessed by only one of the authors, who also gave the anaesthetic. The lack of difference between the groups refers only to the demographic data.

Validation of an observer's assessment of a patient's responses to the clinical situation and premedication, though difficult, has been achieved.[1] The observer's opinion is the most common assessment made of the effects of premedication, particularly in the immediate period before induction of anaesthesia. Observer assessment is normally used in children, and has been successfully used in combination with other indices of stress[2] or in patients who, by reason of the surgery, are unable to mark a linear analogue scale.[3]

Faced with a wealth of data and for the sake of brevity, not all the results were included in the tables. Account *was* taken of the patient's own assessment of anxiety, which was found to be significantly reduced in the lorazepam group 60 minutes after pharmacological premedication and at induction in the placebo group. The chance finding that the triazolam group began with significantly lower scores for the self-assessment of anxiety compared to the placebo group, made it difficult to achieve yet more anxiolysis before induction of anaesthesia. At this time the multiple affect adjective check list (MAACL)[1] was also very close to showing a significant difference at the 5% level by the Kolmogorov–Smirnov test in the patient's assessment of anxiety when the triazolam group was compared to the effects of placebo. This trend agrees with the patient's own linear analogue scale assessments. The relative lack of perceived anxiety persisted in the triazolam group throughout the study period, whereas lorazepam resulted in a significant reduction in the initially higher level of perceived anxiety to reach values comparable to those in patients who had received triazolam. Values for anxiety in both drug treatment groups were consistently lower than for those who received placebo. The triazolam group were also subjectively less alert or clear-headed after 60 minutes and at induction than the placebo group and this may have affected the patients' ability to assess their own anxiety. It is a virtually inescapable concomitant of anxiolytic therapy that sedation results!

The data for the patient's assessment of sleepiness in the anaesthetic room are also available, and show no significant difference between treatment groups, although those who received triazolam were significantly more sleepy at 60 minutes than those given placebo. By 6 hours after the operation, the patient's assessments of anxiety, alertness and clear-headedness were comparable between triazolam and placebo, whereas a significant reduction in alertness persisted in those given lorazepam, which is in agreement with the results of the physician's assessments.

We suspect that the philosophical point whether premedication is given to satisfy the patient or the anaesthetist, will be debated as long as patients continue to present for anaesthesia. For some anaesthetists, sedation of the patient enhances the ability to produce a smooth induction which does not disrupt the anaesthetist, surgeon or other theatre staff. The growing acceptance of the practice of omitting sedative premedication for day cases may erode this view but, if the patients need anxiolysis and the anaesthetist requires sedated patients who recover swiftly, triazolam may fulfill those objectives with the slight penalty of increased dizziness at the time of induction.

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Relative potency of propofol and thiopentone

A meaningful comparison of the relative respiratory depressant effects of propofol and thiopentone (*Anaesthesia* 1986; **41**: 816–20) depends upon a correct assessment of the relative potency of the two agents, so that appropriate equipotent doses can be studied.

Dr Taylor and his colleagues used a potency ratio of 2.5:4 (propofol:thiopentone) which was derived from a study in which the dose of each drug required to produce unconsciousness in 30%, 50% and 80% of patients was calculated.[1] In clinical practice, however, we are generally interested in the dose of induction agent which successfully induces anaesthesia in 95–100% of patients and the effects that this dose may have on respiratory function. It may not necessarily be the case that a perceived equipotency ratio at lower doses is the same as that at clinically used doses, since a ceiling effect may occur with one or other agent. In addition, equipotency was defined in that study against a single endpoint, namely, cessation of counting. This may not necessarily reflect other indicators of depth of anaesthesia such as loss of eyelash reflex or lack of movement on surgical incision, where a different potency ratio may exist.

Using a titrated, incremental administration of propofol to unpremedicated patients, Dundee and his colleagues[2] found a mean induction dose of 1.97 mg/kg in female and 2.08 mg/kg in male patients under 60 years of age. Using similar methods of administration of thiopentone in comparable subjects, the mean dose required to induce anaesthesia has been reported[3,4] to be 4.01 and 5.58 mg/kg. These studies suggest a potency ratio between propofol and thiopentone of 1:2–2.5.

The dose of propofol required to induce anaesthesia in 95% or more of healthy, unpremedicated patients varies in different studies[2,5,6] between 2 and 2.5 mg/kg. In a comparative study of induction of anaesthesia with propofol, methohexitone and thiopentone,[7] we successfully induced anaesthesia in only 17 out of 20 patients (85%) given thiopentone 5 mg/kg, while Taylor and his colleagues induced only 16 out of 20 (80%) with 4 mg/kg. This suggests that the dose of thiopentone required to induce anaesthesia in 95% of healthy, unpremedicated patients is at least 5 mg/kg, and confirms that the potency ratio with propofol is at least 1:2.

Propofol certainly has respiratory depressant properties, but these do appear to be dose-related.[6] Any comparison with thiopentone must be based on the equivalent dose of each agent required for anaesthesia. In our opinion, 2.0 mg/kg of propofol is equipotent with 5 mg/kg of thiopentone.

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We read this paper with interest. The typical trace of tidal volume against time presented, clearly demonstrates that respiratory depression occurred following induction of anaesthesia.

We would like to comment on the method of measurement which has limitations in terms of accuracy, the degree of which is not presented by the authors. Calibration of a pneumotachograph must be performed within the range of flows and circumstances proposed for measurement,[1] including the geometry of connecting tubing and with the gas concentrations to be encountered.[2] The authors do not indicate whether these factors were taken into account.

The technique used is adequate for showing whether or not respiratory depression is occurring by subjective consideration of the trace of volume against time, but not in absolute terms, for the reasons given above. We appreciate that accurate measurement of ventilation at induction of anaesthesia is difficult. Is this one opportunity, perhaps the only one, in an anaesthetic situation where a Respitrace or similar device could be used for accurate measurement?

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A reply

We agree with Dr Grant that any meaningful comparison of all the effects of intravenous anaesthetic agents must be based on equipotency. Thus, we would like to answer some of the specific points Dr Grant has raised concerning equipotency studies.

In our original study comparing the relative potency of thiopentone and propofol,[1] we deliberately used an established technique[2] for determining equipotency. The statistical method of probit transformation is based on a sigmoid dose–response curve. The points on this curve that can be most consistently and accurately found are those on the straight part of the curve, that is, between approximately 30 and 80% of the dose which produces maximum response. As Dr Grant quite rightly points out, as anaesthetists we are most interested in the dose which produces between 95 and 100% response. The point of this technique is to determine accurately what is the 95% response dose. Probit transformation implicitly requires a single absolute defined endpoint. Dr Grant inaccurately states that our endpoint was defined as cessation of counting. Our endpoint was, in fact, the same endpoint as used in the earlier study by Stella *et al.*,[2] namely, response to a direct command, 'open your eyes'. Whether this was the most suitable endpoint is debatable, but other authors[3] consider that loss of eyelash reflex is the least reliable method of determining anaesthesia for patients induced with propofol. As to the non-movement on surgical incision, we agree that a different potency ratio may exist there, but it is not relevant in either of these studies because, at the time of the studies, none of the patients had been submitted to any surgically induced stress.

With regard to the relative potencies of thiopentone and propofol obtained using the technique described by Dundee and his colleagues,[4] this is a valid method for determination of the dose required to induce anaesthesia but, as Professor Dundee states,[4] strictly speaking his paper does not report a study of the induction dose of thiopentone but rather total incremental doses given by a standard method until a predetermined endpoint is reached. It is difficult to accept a figure of 5.58 mg/kg of thiopentone as the induction dose, when the latter refers to a study of the cardiovascular effects of induction of anaesthesia by Seltzer *et al.*[5] in which thiopentone was given in incremental doses of 50 mg every 15 seconds until the eyelash reflex was lost. Furthermore, Thomas[6] has shown that the ED_{95} of thiopentone could be as low as 2.45 mg/kg. It is also difficult to see how Dr Grant arrived at his figure of 2.0–2.5 mg/kg being the dose of propofol that will ensure anaesthesia in 95% of unpremedicated patients. He quotes Cummings *et al.*[3] who state that with 2.0 mg/kg, only 87% of patients went to sleep. Furthermore, Rolly *et al.*[7] found that 2 mg/kg propofol given over 60 seconds induced anaesthesia only in 90% of their patients. In patients under the age of

60 years, Dundee *et al.*[8] found that a dose of 2.5 mg/kg was needed to induce anaesthesia in all patients.

We are also grateful for Drs O'Connor and Wilkinson's comments. We are aware of the various problems associated with the use of the pneumotachograph. We did, in fact, take all these factors into consideration when the pneumotachograph was calibrated in the breathing system used for this study. These problems are well known so we did not elaborate them in detail. We are in total agreement with their comments concerning the use of a Respitrace in this situation, and a study using this apparatus during induction of anaesthesia with thiopentone and propofol, has just been completed.

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Lorazepam in a fast dissolving dose form as a premedicant for children

We read with interest the letter from Dr van den Berg (*Anaesthesia* 1986; 41: 330–1) about the effects of high doses of lorazepam in children, since we have recently had the opportunity to conduct a pilot study to evaluate this drug in a new, fast dissolving dose formulation (FDDF), which was kindly supplied to us by Dr T.V.A. Harry of Wyeth Pharmaceuticals.

The FDDF consists of a wafer which disperses almost instantaneously on contact with the tongue. It is tasteless and, when used as a vehicle for temazepam, has been found to be an effective and acceptable formulation for paediatric premedication[1] conferring advantages over conventional oral preparations which may have an unpleasant taste and whose absorption may be unpredictable.

Fourteen ASA grade I children aged 4–8 years were studied. Each was scheduled to undergo elective ENT surgery as an inpatient. All patients were seen for assessment the day before surgery when parental consent was obtained. The study was approved by the hospital ethical review committee.

The children were premedicated at a set time, 08:00 hours for morning lists and 13:00 hours for afternoon lists. Lorazepam FDDF was given in a dose of 0.5 mg for children who weighed 15–20 kg and 1 mg for children who weighed 20–30 kg (mean dose 0.04 mg/kg, range 0.026–0.05 mg/kg).

On arrival in the anaesthetic room the demeanour of the child was assessed and he was asked about the acceptability of the premedication. His response to venepuncture was noted.

Anaesthesia was induced intravenously with thiopentone 5 mg/kg with atropine 20 μg/kg added. Atracurium 0.5 mg/kg was given and the patient's trachea was intubated with a plain orotracheal tube. Anaesthesia was maintained by manual ventilation of the lungs with a mixture of 66% nitrous oxide in oxygen and increments of atracurium were given as required. All patients received an intramuscular injection of pethidine 1 mg/kg within 5 minutes of induction of anaesthesia.

the end of the procedure with a mixture of neostigmine and atropine, the patients were extubated and transferred to the recovery room. The time taken to respond to name and the incidence of restlessness were noted before the children returned to the ward.

Postoperative assessment on the day of operation comprised three visits at 2-hourly intervals by one of the investigators when restlessness, pallor, vomiting and analgesic requirements were noted. The patients were also seen on the first postoperative day when recall for perioperative events was sought.

The results are tabulated in Table 1. An overall satisfactory result as judged by demeanour in the anaesthetic room, appropriate level of sedation and lack of side effects, was achieved in four patients (27%).

Three patients had delusions after lorazepam and before induction of anaesthesia; these symptoms were transient, but nonetheless gave rise to concern. One patient complained of diplopia and one experienced vivid nightmares prior to transfer to theatre.

Five patients were sedated for a long time (24–36 hours) and three of these were able to recall perioperative events. The parents of these children found this

Table 1. Results of premedication with lorazepam FDDF in 14 children.

Number of patients	Dose (mg/kg)	Effect
4	0.036–0.05	Satisfactory
3	0.03–0.04	Amnesic for induction of anaesthesia
3	0.04–0.05	Delusions
5	0.026–0.048	Prolonged sedation
1	0.04	Nightmares
1	0.04	Diplopia

degree of sedation unacceptable, especially when they were compared with children in the same ward who had received conventional intramuscular premedication. Doses of lorazepam given to these five children ranged from 0.026 to 0.048 mg/kg.

All patients responded to name within 5 minutes of extubation and none was required to stay in the recovery room for more than 20 minutes.

Only three patients appeared to be amnesic for events before induction of anaesthesia; the others were able accurately to recount the form of premedication and method of induction of anaesthesia. Those patients who had recall, all found FDDF lorazepam to be pleasant and palatable.

We therefore found FDDF lorazepam at this dosage to be unpredictable in its effects, and the unacceptably high level of sedation meant that we no longer felt justified in continuing the study. The doses used were comparable with other studies in children of lorazepam in tablet[2] and syrup form[3] in which delusions and prolonged sedation were not found to pose significant problems. We feel that more information about the absorption and pharmacokinetics of lorazepam in this form is required before further study.

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Transnasal anaesthesia of the pharynx, larynx and trachea

Instillation of local anaesthesic into the trachea by cricothyroid puncture is done routinely for awake blind nasal and fibreoptic intubations when conditions warrant. In our institution patients occasionally object to a needle in the neck. We avoid this by using a simple technique which is safe for the patient and which also avoids occasional vocal cord damage and other complications of cricothyroid puncture.

The nasal mucosa is first anaesthetised with cocaine or neosynephrine local anaesthetic combination, and then a soft, well lubricated (Bardex) nasal airway is introduced. This dilates the nasal passage[1] for the subsequent introduction of the tracheal tube as well as providing a route for instillation of local anaesthetic into the pharynx, larynx and trachea. The nasal airway is positioned so that maximum breath sounds are audible. Once this is done, the patient is asked to close his mouth and the opposite nostril is closed either by the anaesthetist's finger or by a plug in the nostril, of a surgical cottonoid soaked in local anaesthetic. The patient is asked to take deep breaths and, on the second deep breath at the moment of peak inspiration, 3 ml lignocaine 4% is injected into the nasal airway. The peak flow carries the local anaesthetic particles to the pharynx, larynx and the trachea.

We achieve good local anaesthesia using this technique, and patients do not react to the introduction of the fibreoptic laryngoscope or tracheal tube. The same could be achieved by the introduction of the fibreoptic scope up to the tip of the nasal airway and injection of the local anaesthetic through the suction port of the laryngoscope while the patient is taking deep breaths. We have done this for several years, and have found that it is easy to master.

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Reference

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Nebulised adrenaline

We read with interest the report on the treatment of croup using nebulised adrenaline (*Anaesthesia* 1986; **41:** 923–6) and would like to take this opportunity to describe a case in which the same treatment was used to alleviate respiratory obstruction of a different cause.

A 4-week-old baby presented for direct laryngoscopy to confirm a diagnosis of congenital laryngeal stridor. Routine anaesthesia enabled direct laryngoscopy and tracheoscopy to be performed with a Stortz paediatric bronchoscope and the diagnosis was confirmed. The baby was transferred to the high care unit for overnight observation.

The baby developed gradually worsening respiratory obstruction over the next 4 hours, shown by severe subcostal and intercostal recession. The degree of stridor reduced and the respiratory rate increased from 50 to 70 breaths/minute. The baby was not cyanosed.

It was felt that the most likely cause of the problem was laryngeal oedema secondary to instrumentation and, rather than attempt re-intubation immediately, it was decided to try nebulised adrenaline. With continuous ECG monitoring, 1 ml of 1:2000 adrenaline (0.5 ml 1:1000 adrenaline diluted with 0.5 ml normal saline) was nebulised with 100% oxygen and given via a fenestrated facemask.

The degree of recession decreased considerably within 5 minutes; the respiratory rate decreased to 50 breaths/minute and the stridor returned to pre-surgical levels.

The baby settled and was soon asleep. The heart rate increased from 160 to 210 beats/minute during the administration of adrenaline but settled after the adrenaline was given. The baby spent an uneventful night with no further respiratory distress and was eventually discharged home 3 days later.

We considered that the development of subglottic oedema, as in croup, was responsible for the reduction of the stridor and for the increase in recession, by a reduction in inspiratory air flow. The administration of nebulised adrenaline has been advocated in the management of postextubation stridor,[1] presumably for the reduction in subglottic oedema.

A tachycardia developed during administration of the adrenaline and it is recommended that treatment should be curtailed if the heart rate exceeds 200 beats/minute.[2] However, infants of the age of our patient tolerate rates in excess of 200 beats/minute better than older children at the age when most susceptible to croup. No dysrhythmia was seen.

We consider that the use of adrenaline in this case prevented re-intubation with all its attendant problems when carried out in the presence of upper airway obstruction, and this treatment should be considered in similar cases. However, it should not be undertaken without the full facilities to carry out tracheal intubation, should the adrenaline fail, by an experienced anaesthetist.

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The case report by Drs Remington and Meakin (*Anaesthesia* 1986; **41:** 923–36) about the use of nebulised adrenaline 1:1000 in the treatment of croup,[1] prompts me to describe a case of acute upper airway obstruction which was managed successfully with a much smaller dose of nebulised non-racemic adrenaline 1:1000 BP.

A 5-year-old boy was admitted with increasing stridor of 3 hours' duration after awakening with a hoarse cough and stridor which had commenced 24 hours earlier; otherwise, the patient had been well. He was apyrexial. In the past 4 years he had suffered from recurrent attacks of croup but these had not required admission to hospital. He had undergone a surgical procedure for congential tracheomalacia at the age of one year.

He was managed at first on the paediatric ward with humidified oxygen and mist. Three hours later he was transferred to the intensive care unit because he was tired, drowsy and was slightly cyanosed, despite oxygen therapy. He had a marked inspiratory and expiratory stridor with a barking cough. The respiratory rate was 45 breaths/minute and breath sounds were delayed, with marked sternal and subcostal recession. Intravenous hydration was commenced and 2 ml adrenaline 1:1000 made up to 5 ml with normal saline was given via a disposable nebuliser attached to a fenestrated mask with an oxygen flow of 5 litres/minute. His condition improved dramatically within 10 minutes and the stridor disappeared completely. His respiratory rate settled to 26 breaths/minute and the heart rate decreased from 150 to 110 beats/minute; he felt greatly relieved and was able to sleep without sedation. The Downes[2] system of scoring of the severity of croup showed that his pre- and post-nebulisation scores were 9 and 4, respectively. He needed another dose of nebulised adrenaline an hour later and thereafter six more doses at approximately 2-hourly intervals. He was observed in the intensive care unit for a further 12 hours and then literally walked out of the unit.

One agrees with the author's view that initial treatment should be conservative, with mist and oxygen therapy and intravenous hydration. However, in our patient a much smaller dose of adrenaline 1:1000 was very effective. In fact, when air entry improves, an even smaller dose may perhaps prove equally effective.

The same treatment was used with dramatic results in a case of stridor after tracheal extubation in a 8-year-old child who had received intermittent positive pressure ventilation of the lungs for 3 days. This very effective, safe and simple form of treatment should find wider application, as very little of the nebulised adrenaline actually enters the trachea, and it may avoid the need for tracheal intubation in both infective and non-infective causes of acute upper airway obstruction.

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Further to the case report by Dr Remington and Dr Meakin (*Anaesthesia* 1986; **41**: 923–6) I would like to report the successful use of nebulised 1:1000 adrenaline in the treatment of post-intubation croup.

The patient was a 10-kg, 17-month-old girl, who was admitted with epiglottitis and whose trachea was intubated uneventfully with a 4.5 mm nasotracheal tube. She was treated with intravenous chloramphenicol. The organism *Haemophilus influenzae* was grown from blood culture. Her temperature had returned to normal on the fifth day of intubation but there was still no leak around the tracheal tube so the patient was anaesthetised and the epiglottis examined and found to have returned to normal. She was therefore extubated and, following extubation, was breathing easily with good air entry to both lungs, no stridor and no sternal or intercostal retractions.

However, after one and a half hours, the patient's condition had deteriorated and her breathing was laboured with severe sternal retraction, intercostal retraction, cyanosis in air and inspiratory stridor. A diagnosis of post-intubation subglottic oedema was assumed.

Preparations were made for re-intubation. The patient was monitored meanwhile with an ECG and a pulse oximeter and oxygen 100% was administered via a nebuliser which contained 3 ml standard l-adrenaline 1:1000. The patient's condition began to improve immediately. The retractions were no longer present after 15 minutes and the stridor had gone. Air entry to both lungs was good and the breath sounds were clear. Oxygen saturation, which had been 92% on 100% oxygen before the administration of adrenaline, had increased to 100%. Heart rate, which had been over 140 beats/minute before the adrenaline, actually decreased during its administration to 120–125 beats/minute.

In view of the patient's improvement she was not re-intubated but was observed closely in the ICU. She was given humidified 30% oxygen to brea
dexamethasone was administered intravenou
condition again deteriorated two and a half ho
the first dose of adrenaline so a further dose
nebulised adrenaline 1:1000 was given and he
tion again improved within 20 minutes. No
episodes of respiratory difficulty occurred
patient made an otherwise uneventful recover

Adrenaline mists and weak topical solu
adrenaline are used in some neonatal units to
or treat post-intubation croup, but there still
be some resistance to its more widespread use
children in this country. In North America
adrenaline has been used in the treatment of b
and post-intubation croup for over 20 years
was pointed out by Dr Remington and Dr
racemic adrenaline would appear to have no
tage over standard 1:1000 L-adrenaline.

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A reply

We welcome these letters which recount exp
with nebulised adrenaline in the treatment of r
croup and postintubation/postinstrumentation
It is interesting that two authors describe a re
in the heart rate following nebulised adrenaline
of severe croup, presumably the result of in
ventilation. Although excellent results were c
in all these cases with reduced doses of ad
1:1000, two of the patients were in fact quite
reduced dose may be appropriate for very
children but we still recommend a dose of 5
children aged over one year and weighing mc
10 kg. We have used this dose of adrenaline suc
in five more children with infective croup si
original case report; the youngest was aged 18
There was marked clinical improvement in ea
and no patient developed significant tachyca
dysrhythmia.

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Calories in sedation

Little interest has been shown in the calorific content of the current formulation of propofol (Diprivan). The drug is emulsified in 10% Intralipid. Consequently, a patient in an intensive care unit who is sedated by an infusion of propofol at the rate of 20 ml/hour, would receive 528 kilocalories per day. This is 25% of the average daily calorific requirement, and may be as much as 50% of the daily amount of Intralipid in an average intravenous feeding regimen.

In these days of a straitened Health Service
of dual function preparations should be encou

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Spinal anaesthesia for Caesarean section

The introduction to the study of hyperbaric lignocaine (*Anaesthesia* 1986; **41:** 906–9) refers to the high blocks obtained with isobaric bupivacaine and, thus, its unsuitability for operative delivery in obstetrics, particularly after failed intubation. We agree with the findings of the few reports of the use of plain 0.5% bupivacaine for spinal Caesarean section,[1–3] that the spread can on occasions be high and unpredictable but Russell's case[4] which is quoted was of a total spinal using 2 ml of 0.75% bupivacaine. We wish to report our own experience of the use of plain 0.5% bupivacaine for spinal Caesarean section to give some perspective.

In this hospital's obstetric unit approximately 250 women have received spinal anaesthesia for Caesarean section with 0.5% plain bupivacaine during the past 2 years. This has become our most commonly used method of regional anaesthesia for elective Caesarean section, and has remained popular because of the rapid onset of the block and the quality of analgesia produced for surgery. The need for supplementary analgesic drugs at any stage during the operation has been rare. High levels of sensory block have occurred and a number of women have reported paraesthesiae in their hands. However, the high levels of block, which tend to be sensory rather than motor, do not cause difficulties with respiration and recede rapidly. In only one patient has the level of block caused concern, when for 5 minutes she had difficulty in swallowing her saliva and required oral suction.

Injection is made in the left lateral position using a 26-gauge needle and the volume of 0.5% bupivacaine varies between 2.5 and 3.5 ml depending on the patient's height. She is turned to the supine wedged position which encourages earlier spread[2] and minimises further extension of the block when she is moved into theatre.

Hypotension occurs with any form of subarachnoid analgesia in the obstetric patient, except perhaps a saddle block, unless care is taken to avoid it by preload with intravenous fluids and vasopressors as required. It is not a technique for the inexperienced operator-anaesthetist or for those with inadequate assistance. Headache is low in both incidence and severity; were it otherwise, nursing staff and obstetricians would have encouraged us to stop the technique. However, we feel that one disadvantage of subarachnoid rather than epidural bupivacaine, is the rate at which pain increases once the block begins to recede. Epidural block wears off more gradually and allows the opportunity to anticipate the pain with intramuscular papaveretum, whereas adequate pain control after subarachnoid block in the immediate postoperative period has proved difficult on occasions. This is the subject of further investigation.

In conclusion, we do not wish to champion the use of 0.5% plain bupivacaine for spinal anaesthesia for Caesarean section but, since we have found something that works well for us, we have chosen to stick with it.

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Angioneurotic oedema following etomidate/lignocaine

Etomidate has been described, with some authority, as an 'immunologically safe' anaesthetic agent,[1] and allergic reactions to lignocaine are sufficiently rare for some authors to dispute their existence at all,[2] although a reaction which was confirmed by antibody studies was reported in 1981.[3] We should like to describe a case of angioneurotic oedema following the intravenous injection of a mixture of those agents.

The patient was a 19-year-old, 50-kg girl, who presented as a day case for a minor orthopaedic procedure. She was a healthy non-smoker with an unremarkable medical history, whose only medication was an oral contraceptive.

She received no premedication and in the anaesthetic room appeared to be mildly anxious. Prior to induction 1 ml lignocaine 1% plain (preservative-free) was added to 20 mg etomidate in propylene glycol 35% (2 mg/ml); a technique which has been reported to attenuate the pain on injection associated with etomidate.[4] Anaesthesia was induced by the slow intravenous injection of 7.5 ml of this solution (15 mg etomidate; 7.5 mg lignocaine). Several forceful sneezes occurred before the patient began to breathe normally 67% nitrous oxide in oxygen, together with halothane, via a Mapleson A system with a rubber facemask. She showed signs of increasing respiratory difficulty within 3–4 minutes and complete respiratory obstruction followed; this was relieved by tracheal intubation facilitated by suxamethonium 50 mg. The patient had by this stage started to develop peri-orbital oedema, although there was no obvious palatal or epiglottic swelling. The laryngeal inlet was never seen. A precordial stethoscope and blood pressure cuff had been in place throughout: there was no auscultatory evidence of bronchoconstriction and there were no gross changes in the pulse rate or blood pressure. The decision was made, when the airway was secure and the cardiovascular system stable, to continue with surgery and the remainder of the anaesthetic was uneventful. Tracheal extubation was performed cautiously in the recovery area: laryngoscopy revealed some palatal and uvular oedema but there was minimal inspiratory stridor. The peri-orbital oedema persisted for more than 24 hours but otherwise the patient made a complete recovery.

Early blood samples showed 30% complement C_3 conversion, a normal IgE of 50 IU/ml and an elevated IgM of 2 g/litre which, together with acute phase increases in complement levels, suggested a pre-existing immune response to subclinical infection. Six weeks later a bronchial provocation challenge with histamine was negative, after which intradermal testing was performed. The patient showed a positive response to the intradermal injection of 0.1 ml lignocaine 1% plain (preservative-free), although there had been no reaction at higher dilutions. The validity of intradermal testing using local anaesthetics has been questioned,[5] unless the test is part of a progressive challenge in the protocol recommended by deShazo and Nelson.[6] Nonetheless, the immunologist who carried out the tests was satisfied that they demonstrated hypersensitivity to lignocaine and did not feel that further intradermal testing with etomidate would be useful. More complex investigations such as assay of lignocaine antibodies, were unavailable.

It is possible that this patient was sensitive to lignocaine, but hers was not an IgE-mediated allergic reaction and we must remain uncertain as to the precise cause of the angioneurotic oedema. Contributory factors may, however, have included a pre-existing alteration in immune status due to infection, stress provoked by anxiety, and a synergistic reaction between etomidate and lignocaine. This last aspect may attain wider significance if there is greater use of the propofol/lignocaine mixture which has recently been shown to reduce the pain on injection associated with that induction agent.[7]

This case illustrates that various factors may conspire to make it difficult to identify the exact cause of an anaphylactoid reaction. Of much more importance, however, is the fact that such reactions remain largely unpredictable, and that when anaesthetists administer intravenous agents they can never afford to be blasé, however blameless may be the reputations of the drugs.

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Ventilation and the oculocardiac reflex

We are concerned about some of the methods used by Mirakhur, Shepherd and Jones (*Anaesthesia* 1986; **41**: 825–8).

We think that when one wishes to study the role of controlled ventilation and glycopyrronium in the incidence of the oculocardiac reflex (OCR), every attempt should be made to ensure that all other factors are equal and that any differences which are observed are likely to be the result of the type of ventilation and of the anticholinergic used. The experimental design does not meet these basic requirements. In fact, induction of anaesthesia was not standardised since it was achieved either with intravenous thiopentone (in dosages changing from 5 to 6 mg/kg) or by inhalation of nitrous oxide/oxygen and halothane (in percentages not mentioned). Maintenance of anaesthesia was not standardised because it was accomplished with halothane in a very wide range of percentages (1–2%). Groups were not comparable because 50 patients (groups 1 and 2) received suxamethonium for tracheal intubation while 50 other patients (groups 3 and 4) received vecuronium for intubation and controlled ventilation of their lungs. It is known that vecuronium may facilitate bradycardia and cardiac dysrhythmias[2,3] but the contribution of vecuronium to the results of the study was not addressed in the conclusions of Mirakhur and his colleagues. Moreover, if one considers that every child was premedicated with oral trimeprazine (2 mg/kg) and that all children received intravenous pethidine (0.5 mg/kg), the possibility of different planes of anaesthesia and of different pharmacological interactions has to be considered.

Another major concern arises from the way we were cited. In 1983, we described a method for the detection and recording of the OCR.[4] Using a simultaneous recording of the pulling force applied to the extra-ocular muscles, and of the heart rate, we demonstrated that the type of stimulus clearly influences the incidence of the OCR: abrupt and sustained traction (square-wave or type A stimulus) was more reflexogenic. We emphasise that Mirakhur *et al.* did not standardise the type and the intensity of the pulling force applied to each extra-ocular muscle but also, we submit that it is possible to diagnose a positive OCR before an important and potentially dangerous decrease in heart rate of 20% or more and/or the development of cardiac dysrhythmias.

Finally, the study by Mirakhur *et al.* introduces questions concerning the experimental design and the conclusions drawn from the results obtained. Why so different techniques of anaesthesia and so different dosages of anaesthetics? Why not use controlled and well-defined stimuli? How many surgeons operated on the 100 children in the study? Given that stimuli were not standardised, how many medial recti were included in each group? How much time was allowed between tracheal intubation and the first extra-ocular traction, in each group? How much time was allowed between the intravenous injection of pethidine and the first muscular traction, in each group? Why adopt less than 70 beats/minute to release the muscular traction in children whose ages vary from 1 to 14 years? Is it right to consider that the haemodynamic consequences of such a sudden and deep bradycardia are identical in children 1 to 14 years old? Is it acceptable to expose 50 children (groups 2 and 4) to a significant risk of sinus arrest? Was or was not an informed consent obtained from the parents?

No one would be able to reproduce this study because it lacks too much essential information and is insufficiently standardised.

We certainly admit that vecuronium is not a good choice for the prevention of the OCR. This is particularly true if no anticholinergic drug is given and when surgeons are unable to avoid precipitate and aggressive manipulation of the extra-ocular muscles.

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A reply

Thank you for the opportunity to reply to Dr Blanc's letter.

May we first of all point out that our study was carried out in a real clinical setting and not from a theoretical standpoint. Contrary to Dr Blanc's assertion, the four groups in the study were well matched and comparable, even with regard to the method of induction and maintenance of anaesthesia (Table 1 in the original paper). The intravenous or inhalational methods of induction and the concentrations of halothane used, were based on clinical considerations and to have done otherwise would have resulted in a study with very little relevance to usual clinical practice. The oculocardiac reflex is a specific response to traction on the extra-ocular muscles and occurs irrespective of the induction technique or muscle relaxant employed. Vecuronium, because of its minimal cardiovascular effects, was considered to be a very suitable relaxant for the children who were ventilated in our study. Indeed, vecuronium was *specifically* chosen for its lack of antimuscarinic effects which could conceivably modify the response to traction on the extra-ocular muscles. In fact, in our study, the choice of muscle relaxant was probably of little relevance, since traction on the extra-ocular muscles was applied more than 10 minutes after relaxant administration and intubation. When vecuronium is used, the heart rate may be lower in the presence of other drugs such as fentanyl, but such a technique was not used in the present study and the heart rate prior to applying traction on the extra-ocular muscles was similar in the comparable groups (i.e. groups 1 and 3, groups 2 and 4). Moreover, to our knowledge, there have not been any reports of bradycardia with the use of vecuronium in children.

We do not understand how the use of the same dose of pre-operative trimeprazine and intra-operative pethidine in all children would result in different planes of anaesthesia and differing degrees of pharmacological interaction. We have used trimeprazine, pethidine and halothane and administered vecuronium to children for the last three years, and have not encountered any untoward pharmacological reactions. Perhaps Dr Blanc might have cared to support his statement relating to these proposed interactions with some evidence.

Dr Blanc is concerned at the way in which we cited their study[1] and, again, this is unjustified. We agree that gentle traction may be associated with a less severe decrease in heart rate than more vigorous traction, but this was not an issue in our study since we were not comparing the type of stimuli. The same surgeon carried out all the operations and he was not instructed to change his operating technique in any way, thus ensuring that the traction applied to the extra-ocular muscles was similar, as far as possible, in all the groups. Again, although traction on the medial rectus muscle is associated with greater decreases in heart rate,[2] it is irrelevant in the context of the present study whether the heart rate decreased because of traction on the medial rectus or any other extra-ocular muscle. We would reiterate that a decrease in heart rate of 7.2 beats/minute is a totally unrealistic clinical criterion—very few anaesthetists would be alarmed by such a fall, whereas they would be concerned if the heart rate fell by 20% or more, or if cardiac dysrhythmias occurred. This was the reason why we fixed a heart rate of 70 beats/minute to intervene during surgery, even though a decrease in heart rate of 20% or more was taken to indicate a positive oculocardiac reflex.

It is astonishing to read that Dr Blanc is concerned at the exposure of 50 children (groups 2 and 4; those who did not receive prophylactic anticholinergic) to the risk of bradycardia when he and his colleagues actually advise against the administration of anticholinergic drugs for the prevention of the oculocardiac reflex.[1] We used well established anaesthetic techniques with full approval from the Regional Ethical Committee. In addition, the patients were routinely monitored and provision was made in the study for taking corrective steps. If Dr Blanc has changed his opinion after reading our and other papers on this subject, it is only for the better. We are satisfied at having brought home the point that anticholinergic drugs provide the proper prophylaxis for the oculocardiac reflex.

Dr Blanc is raising theoretical questions which have no clinical relevance. We carried out a proper, prospective study with well matched groups which has conclusively shown controlled ventilation to be of no value, and anticholinergic drug administration to be the better prophylaxis against the occurrence of clinically significant decreases in heart rate due to traction on the extra-ocular muscles. We feel that Dr Blanc would be better employed carrying out a controlled, prospective study when he would, it is hoped, be able to confirm our results rather than advocating controlled ventilation, but not anticholinergic drugs, for the prevention of the oculocardiac reflex on the basis of a study[1] in which no patient was either ventilated or received prophylactic intravenous anticholinergic drugs. Significant progress in the quality and safety of anaesthesia will not result from following such unproven advice.

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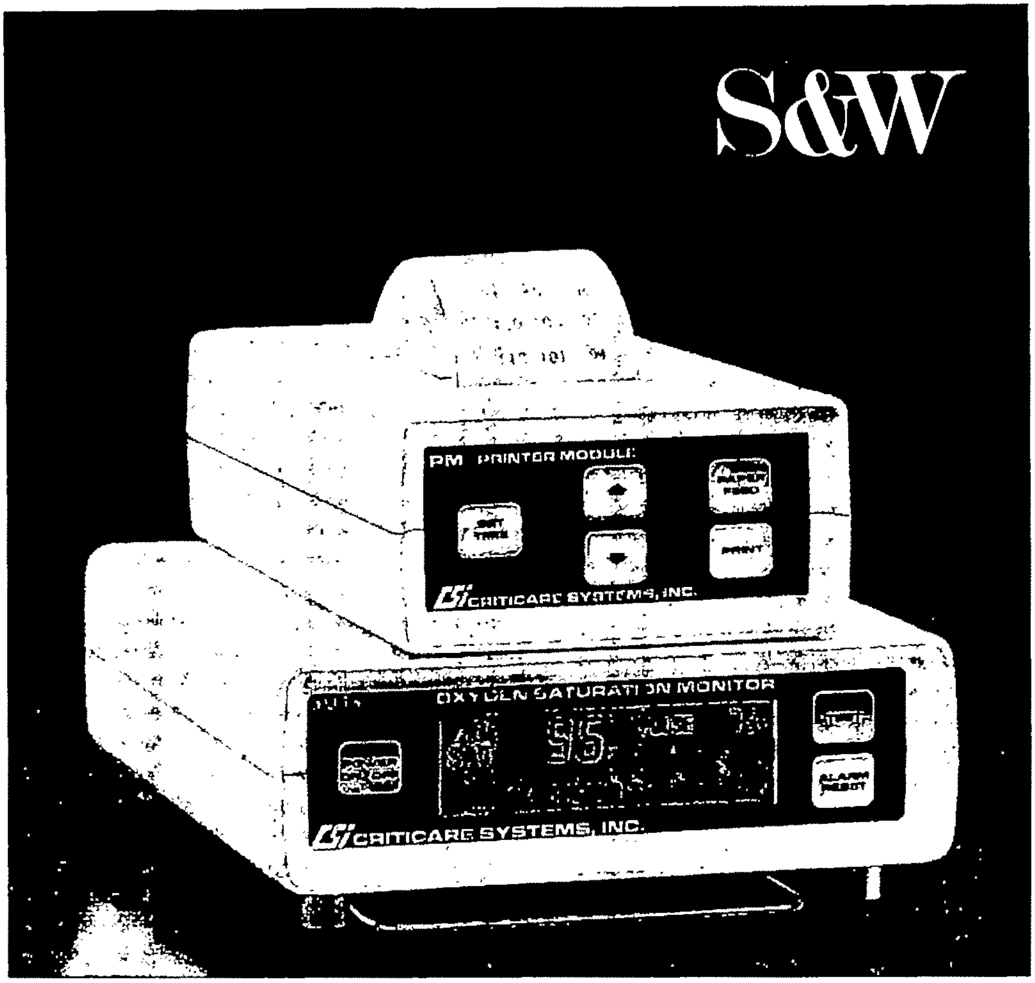
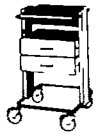
A nerve palsy occurring during the recovery period

It was fascinating to read Dr Leach's letter (*Anaesthesia* 1986; **41:** 762) since I wish to report another case of nerve palsy of the posterior interosseous nerve.

A 28-year-old woman presented for dilatation and currettage with no previous relevant medical history and was given an oral premedication of temazepam 20 mg and promethazine 25 mg. In the anaesthetic room, a 20-gauge cannula was inserted into the dorsum of her right hand (she was left-handed) and 300 mg of 2.5% thiopentone injected, and the patient went to sleep in the normal way. Anaesthesia was then continued with spontaneous ventilation of a mixture of nitrous oxide, oxygen and isoflurane. The patient's arms were positioned across the upper abdomen and padded arm supports were carefully placed in position. A sphygmomanometer cuff was placed on the left arm. The operation took only a few minutes. At the end of the operation, the canvas poles were inserted (the elbows were lifted during this procedure) and the patient was then turned on her right side on the trolley canvas with the elbows bent and her hands near her face. The patient was sleepy and she remained in this position for about 2 hours, and on this occasion the canvas poles remained in position. When the patient was fully awake she could not extend the fingers or thumb of her right hand. The patient was seen by a consultant neurologist who found she had complete inability to extend the fingers due to paralysis of extensor digitorum, extensor digiti minimi and extensor indicis, inability to extend the thumb due to paralysis of extensor pollicis longus and extensor pollicis brevis and, in addition, the patient had extension of the wrist with radial deviation due to paralysis of extensor carpi ulnaris and sparing of extensor carpi radialis longus supplied direct by a branch of the radial nerve arising above the elbow. There was no sensory deficit or other neurological finding and there was no swelling of hand or forearm. The diagnosis was made of posterior interosseous nerve palsy and this was confirmed by nerve conduction studies and electromyography.





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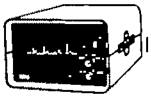

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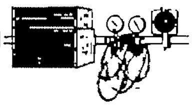

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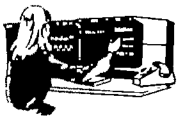

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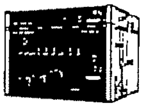

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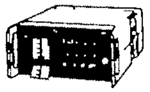

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One month later there was no improvement, so the elbow was explored by a consultant orthopaedic surgeon. It was found that the posterior interosseous nerve was lying close to the neck of the radius protected by an unusually thin layer of supinator muscle that made it vulnerable to light pressure. At operation, direct electrical stimulation of the posterior interosseous nerve produced a brisk response in all the paralysed muscles. Shortly after completion of the operation, the patient had some active extension of the thumb and index finger of the right hand and, in the following weeks, made a full recovery.

It would seem, after a full review of events, that the most likely explanation of the posterior interosseous nerve palsy in this patient was pressure on the elbow by the canvas pole on which she was lying whilst in recovery. Many patients are restless during recovery and they wriggle in such a way that, if a pole were present, it could cause pressure on the elbow; I now remove poles from the canvas as soon as the patient has been transferred to a trolley.

In Dr Leach's case, extensor pollicis longus and extensor indicis were paralysed. These muscles receive their nerve supply high in the forearm[1] and it is difficult to see how even an injection of 18 ml of thiopentone in the dorsum of the hand could extravasate at a site remote from the initial injection; perhaps mechanical pressure at the elbow is a more likely explanation?

This is not a new observation but is worthy of emphasis: if the canvas poles are left in a canvas while the patient is recovering in the lateral position, a nerve palsy can occur.

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D.I. Hughes-Davies

Reference

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Epidural block for Caesarean section

The special article by Crawford, Davis and Lewis (*Anaesthesia* 1986; **41:** 1039–46) was interesting but one was surprised to note that 47% of their patients required more than 150 mg bupivacaine for Caesarean section. The very large volumes (up to 80 ml) of local anaesthetic used in some of their patients, suggest a possible fault in their technique and it is worth considering the reasons for inadequate spread of local anaesthetic.

Studies using contrast media have shown that fluid may move rapidly out of,[1] or into, the epidural space[2] through the intervertebral foramina. In young patients the epidural space is like a sieve and injected fluid rapidly leaks out through the intervertebral foramina. In the fully lateral posture this leakage through the inferiorly placed foramina is facilitated by the effect of gravity, whilst in the supine posture the fluid has to traverse upwards to the now more superiorly placed foramina before leakage may occur.

Spread of local anaesthetic within the potential epidural space should be influenced by the pressure in the space. The epidural pressure is higher in the supine than in the lateral posture[3] and, hence, spread of local anaesthetic should be greater in the supine posture.

Spread of fluid is volume dependent. A large volume spreads further before its leakage out of the space. Grundy *et al.*[4] used single injections of 15 and 20 ml local anaesthetic in the supine posture with a lateral tilt, and achieved adequate analgesia for Caesarean section in all their patients.

An injection technique where the first dose consists of at least 15 ml local anaesthetic given with the patient supine with a lateral tilt, or supine with left uterine displacement, achieves wider spread, rapid onset of analgesia and superior results without the possibility of exceeding the manufacturer's recommended dose.

Dudley Road Hospital,
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J.L. Shah

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A reply

Thank you for the opportunity to reply.

No doubt many faults can be identified in the management of the cases referred to in our report. It would be surprising if that were not so, in a series of about 1000 mothers cared for under routine clinical circumstances, during a period of 15 years. However, apart from a small number of cases, the administration of large volumes of bupivacaine solution is not included in the list of faults.

Frankly, one is not greatly concerned about the total mass of bupivacaine injected (provided that it goes into the right place) but I am, however, greatly concerned about the rate of administration. Injection of more

than 5 ml of solution at any one time is not acceptable. In common with others, I have on several occasions in the past seen a single injection of 10 ml bupivacaine (0.5%) to be sufficient to provide excellent analgesia for the whole operation. A single injection of 15 or 20 ml in such a case would probably have led to a lot of trouble. In the obstetric field, speed is much less important than safety and, if deliberation invo
tendency to administer a higher dose, I have no
about the balance of virtue of the two techniqu

The Birmingham Maternity Hospital, J.S. Crav
Birmingham BT15 2TG

Response rates

A common problem encountered with reasearch follow-up postal questionnaires is their poor response rate. We have recently been surprised to obtain an almost 100% response to a postoperative pain questionnaire.

Previous response rates have averaged around 60% when questionnaires were accompanied by a printed prepaid envelope. However, we now apply a first-class stamp, removed from a book of stamps, t
addressed envelope in the presence of the patient
simple act appears to motivate the patient to
their questionnaire.

St James's University Hospital, S.N. C
Leeds LS9 7TF K.H. S

Patients with problems

It is our practice to leave a conspicuous record as well as to advise the patient whenever any significant problem occurs not, as Dr P.A. Isaac suggests (*Anaesthesia* 1986; **41:** 1158–9), inside the notes but on the front of their file. A prepared stamp previously stated 'Known to be sensitive to . . . .'. We have introduced a specific departmental stamp headed 'Anaesthetic problem' with space for details of its nature and a date referring to records within. This is held available for immediate use in the operating theatre, recovery room or anaesthetic office. Clear identification of possible difficulties has been most helpful. All staff are forewarned and our searches through multiple notes are simplified.

Royal West Sussex Hospital, M.R. Nott
St Richard's,
Chichester PO19 4SE

It was interesting to read the letter from Dr P.A.
(*Anaesthesia* 1986; **41:** 1158–9) which emphasis
importance of recording any problem associate
anaesthesia in a particular patient, especially
regard to airway management or difficult intuba

We have for the past 5 years in this hospital ad
the procedure Dr Isaac recommends. When a pr
arises this is discussed with the patient who is
issued with an 'Anaesthetic difficulty warning'
which summarises the problem; a letter is sent
patient's General Practitioner, a copy of wh
inserted into the casenotes, and the patient's nam
details of the problem encountered, are register
the Anaesthetic Department's computer. We
registered 22 cases of difficult or failed intubation
this procedure was instituted.

Glan Clwyd Hospital, P.M.
Bodelwyddan,
Rhyl,
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Marcain shakes

We noted with interest the letter describing Marcain shakes in association with an obstetric epidural procedure (*Anaesthesia* 1986; **41:** 765). We would like to report a similar episode in a non-obstetric patient who had epidural analgesia.

A 60-year-old female, weighing 71 kg, underwent a laparotomy for severe recurrent pancreatitis. She had angina which was controlled by propranolol. After premedication with diazepam 15 mg, anaesthesia was induced with etomidate 8 mg, vecuronium 5 mg and fentanyl 150 μg. Radial arterial and pulmonary artery flotation cathethers were inserted, and anaesthesi
maintained with supplements of fentanyl and is
ane. The procedure, lasting 5 hours, entailed lig
of a splenic artery aneurysm, splenectomy,
pancreatectomy and cholecystojejunostomy. Sh
subsequently admitted to the intensive care unit,
analgesia was provided by an infusion of bupiva
0.25% without adrenaline, via an epidural cathe
T_{12}–L_1. Twenty-two hours after its insertion, she
to complain of shaking in her right leg and, to a
extent, in her right arm. Axillary temperature

37.4°C, Pao_2 15.9 kPa and $Paco_2$ 5.27 kPa (Fio_2 0.40). Systemic and pulmonary arterial pressures were normal, and the pulmonary artery occlusion pressure was 7 mmHg. The catheter was removed, with relief of her symptoms within 30 minutes. The patient subsequently made an uneventful recovery and was discharged home on the 14th day after operation.

We conclude that postepidural shivering in association with bupivacaine is related neither to pregnancy, to hypoxia, to hypovolaemia nor to hypothermia.

Withington Hospital,
Manchester M20 8LR

A.A.C. Dow
R.G. Wilkins

Narcoanalgesia

We have chosen the combination of regional analgesia and light general anaesthesia as the mainstay of our everyday techniques for the last five years.

A variety of procedures, from upper and lower abdominal surgery to operations on the hip, lower limbs, vertebral column, kidneys and some thoracic operations, have been done using this method, for which we would like to propose the name 'narcoanalgesia'. The only thing new is the name. The idea came from Crile, with his theory of anoci-association, which appeared in 1899.[1]

In the last thousand cases we have managed under narcoanalgesia, the general anaesthetic was by tracheal inhalation anaesthesia with nitrous oxide–oxygen mixtures with or without the addition of low concentrations of halogenated agents. Sometimes fentanyl, in 25–50 μg doses, repeated when deemed necessary, replaced the volatile anaesthetics.

The regional methods were epidural or spinal analgesia, single shot or continuous. A continuous spinal with a nylon catheter threaded through an 18-gauge Tuohy needle gave excellent results in old people and especially in poor risk cases.[2] We are now in the process of evaluating the data on these 1000 cases but we consider that the name 'narcoanalgesia' is worth preliminary consideration.

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A. Fortuna

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Nasal intubation

Following the recent correspondence on the problems involved in nasal intubation, it is surprising that no-one has referred to your own pages where A.G. MacKinnon and M.J. Harrison (*Anaesthesia* 1979; **34:** 910–11) describe the use of a red rubber Jaques catheter to lead the tracheal tube through the nose and nasopharynx.

The use of the technique in our outpatient dental clinic has resulted in less blood loss from the mucosa of the turbinates and fewer inadvertent adenoidectomies, and one has no further worries about causing the offensive nasal discharge all too common in young patients.

More importantly, it enables one to introduce tracheal tubes through passages otherwise closed by bony enlargements and septal deviations, not unusual in handicapped patients; a process often accompanied by much crunching, but rarely bleeding. On a point of economy, we have discovered that the catheters withstand autoclaving well and will last for many months.

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T.B. Webb

Anaesthesia, 1987, Volume 42, pages 330–334

Book reviews

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Handbook of Epidural Anaesthesia and Analgesia

B.G. Covino and D.B. Scott. Pp. 175. Grune and Stratton, Inc., 1985. £41.50.

The aim of this textbook is to provide basic information about epidural anaesthesia and analgesia to the average anaesthetist, and particularly to the anaesthetist in training. The names of both authors command considerable respect in the field of regional anaesthesia, and for this reason alone the book is of great interest. Add the name of Poul Buckhöj as illustrator and the reader knows something special may result.

The text begins with a splendid, clearly illustrated section on the anatomy of the epidural space which provides a wealth of relevant information. The next section, on physiological considerations, begins obliquely by telling the reader where local anaesthetic agents do not work, and then gets round to giving the author's best guess as to where they may have their site of action. This is followed by some redundant information on ascending and descending pathways before a more detailed consideration of other physiological effects of epidural anaesthesia. It may have been helpful to the trainee anaesthetist to emphasise the clinical significance of the observed changes in this section, although this is covered in later sections.

The chapter on pharmacological considerations is a model of clarity and is simple and comprehensive. This reviewer almost wishes that it was not and would have liked to read the author's candid opinions on more controversial topics such as the use of 2-chloroprocaine for surgery, and the use of 0.75% bupivacaine or prilocaine for obstetrics. However, the authors give clear views on the use of 'supercaine', the practice of compounding different local anaesthetic agents in an attempt to provide fast onset and prolonged duration. It is to be hoped that their advice, that there is little scientific basis for this practice, will be heeded. In addition, they are to be congratulated for entering into the therapeutic minefield of epidural opioids. They restrict themselves to fairly general statements but these will provide a firm basis for good clinical practice.

In the section upon technical considerations the dividing line between American and European practice can be observed, and due attention is paid to variations in technique. The descriptions of how to perform the various approaches are excellent although there are a couple of statements with which this reviewer does not agree. Nobody, however eminent, could convince me that 10 ml of local anaesthetic solution deposited at C_7–T_1 would only block from C_4 to T_8. This seems a pretty hefty volume, and reference to the experts found in a well-known university town in the north of Sweden (where I suspect at least one of the authors acquired his experience of this technique), suggests that a smaller dose of 4–5 ml will block from C_2 to T_5 without difficulty. The authors also suggest the use of a conventional long-bevel disposable needle for doing caudals. I think that the use of a short-bevel needle markedly reduces the number of times that blood is encountered with this approach and adds considerably to its reliability.

The authors finish off the book with a rousing chapter on clinical considerations which sells the advantages of epidural anaesthesia to the hilt (or is it hub?), and a final chapter on complications which is to a very high standard. Experienced anaesthetists may like to start with this chapter and then proceed to the rest at their leisure. Pertinent references appear at the end of each chapter and not in the text. Considerable thought must have been given to this decision but I do not think it was the right one. Perhaps the authors feel it will be character-forming for the trainee anaesthetist to have to read all the reference titles and guess which is the appropriate one instead of being able to go straight to the paper of interest.

Overall this is a splendid book; it contains a wealth of basic information with very little that is redundant. It will appeal to experienced anaesthetists as well as to the anaesthetist in training and it is a must for every departmental library. The strength of the text is matched by the superb quality of the illustrations and these will undoubtedly be ruthlessly plundered to demonstrate the principles of epidural anaesthesia in departmental lectures throughout the world.

J.E. Charlton

Intensive Care Medicine

Edited by J.M. Rippe, R.S. Irwin, J.S. Alpert and J.E. Dalen. Pp. 1203. Churchill Livingstone, 1985. £72.90.

It would have been more realistic to have entitled this monumental volume 'Medical Intensive Care', because that is what it is really about. Sixty-three of the 110 contributors, most of whom work in the University of Massachusetts Medical School, are professors of medicine. Indeed, with that assumption of superiority which anaesthetists, paediatricians and surgeons, all in their own ways as likely to be strongly committed to intensive care, find so irritating, the Editors make no bones about this; they begin their preface with the statement that '*Intensive Care Medicine* is in essence advanced medicine'.

The book consists of a large number of monographs on various aspects of intensive care, each written by a specialist in the subject. The style is that of the essay rather than of the didactic text, with extensive references up to 1983, a reflection of the time it takes to produce a book of this magnitude. The first section is devoted to procedures, including most types of vascular access, insertion of tracheal tubes and chest drains, endoscopy, tracheostomy, defibrillation, cardioversion, aortic counterpulsation and lung biopsy, but not of urethral catheterisation, surely as important a subject as arterial puncture. However, the most startling omission from the title list is that of artificial ventilation, which is covered neither in this section nor in the later one on Anesthesiologic issues (which turn out to be the recovery room and the management of pain, anxiety and psychosis). Other sections are devoted to pulmonary, cardiovascular, renal, haematological, endocrine and infectious problems. Coronary care, gastrointestinal disease (including liver failure), overdoses and poisoning, dermatological, rheumatological and immunological problems are the subjects of further sections. There is a short section on surgical problems and a section on neurological problems in which only four of the 61 pages are devoted to head trauma. The final section deals very well with important moral, ethical and legal issues and with public policy in intensive care medicine, although the care of organ donors is not discussed.

What is the place of this book? It is not meant primarily for the trainee anaesthetist, surgeon or paediatrician, although it will be of use to those who have chosen intensive care as their special interest. This is essentially a reference volume which will find its natural place in the library, rather than on the desk in the intensive care unit. It deals very well with the subjects it addresses, and complements the many therapeutically oriented texts available.

J.V. Farman

Cardiopulmonary Bypass

Edited by K.M. Taylor. Pp. 439. Chapman and Hall, 1986. £37.50.

This is a comprehensive review of cardiopulmonary bypass, its physiology (and pathophysiology!), its technical aspects and its problems. There is hardly an aspect of this procedure that is not examined in informative detail with many helpful illustrations. There are also chapters on anaesthesia, myocardial protection and counterpulsation. Each chapter is accompanied by a helpful list of references.

The 23 contributors are drawn both from the UK and the USA and many are established authorities. Personally, one would have liked a special chapter on brain damage since recent work has emphasised the frequency of this disturbing complication and Sotaniemi and his colleagues have now shown that long-term cerebral damage from prolonged bypass may not be detectable for 5 years (*Stroke* 1986; **17:** 410–16). Pearson's chapter on micro-embolism certainly merits study in this context. The problem of myocardial protection has not yet been resolved, and heart failure remains a major complication of open heart surgery. Despite Braimbridge's enthusiasm for cold cardioplegia, many surgeons have abandoned this technique in favour of intermittent aortic clamping and whilst he states that 99% of USA centres still use cardioplegia, this is certainly not the figure for the UK. Today in many (? most) centres, technicians run the pump virtually unsupervised by a medical practitioner. The medicolegal aspects of this situation have not yet been fully explored, but whilst no great depth of knowledge is required to undertake this responsible task, this book reminds us of the wealth of information in this field. One doubts if anyone engaged in open heart surgery is sufficiently knowledgeable not to benefit from dipping into this important work and it should certainly be in every cardiac surgical unit, and kept there!

A. Gilston

Epidemiology in Anaesthesia

Edited by J.N. Lunn. Pp. 174. Edward Arnold, 1986. £16.75.

Until I read this book I imagined the title would refer to such topical events as the transmission of AIDS via anaesthetic apparatus, and I was very pleasantly surprised to find that each chapter conveyed an interesting and useful concept.

The authors themselves defined their subject in different ways but the definition used in Chapter 4 is probably the most apt, namely, 'the study of the distribution of a disease or a physiological condition in human populations and of the factors which influence this distribution'.

Subjects covered range from the history of epidemiology as applied to anaesthesia, through statistical methods and their application, to specific clinical problems. Reading each chapter leaves one with a most healthy scepticism about the value of a great deal of published research. This in itself makes the book essential reading for all thoughtful anaesthetists.

There are 10 chapters and each one is written so that a non-specialist can understand quite complex concepts. Most of the major problems which have arisen in anaesthesia since its introduction into medicine are described. These include mortality, morbidity and risk studies and the questions of halothane hepatitis and operating room pollution. There is a very useful chapter entitled Strategies for the Development of New Anaesthetic Drugs. This almost makes one feel sorry for the pharmaceutical companies by pointing out the problems they are faced with before they can even begin to release a new substance.

The last three chapters are different approaches to the same topic, namely, anaesthetic record keeping and documentation. Those of us who have been involved in the *Confidential Enquiry into Peri-operative Deaths* have recognised how totally inadequate most standard anaesthetic forms are but even more, how remiss we anaesthetists can be at completing even these paltry records. The Cardiff system clearly has a lot to recommend it but even so, the extraction of data from clinical records is notoriously difficult and Dr Seed's chapter on documentation confirms this.

Doctors Bushman and Cushman describe the application of computers to anaesthetic record keeping. Their chapter starts off quite optimistically but, unfortunately, problems beset every solution they propose. It seems inevitable and desirable that some form of standardisation must come but for the foreseeable future it will almost certainly have to be a variant of the pen and ink record rather than the light-pen or keyboard. A medicolegal action which is pending demonstrates that the police and the legal profession are prepared to question the accuracy of anaesthetic records. If nothing else this should make us all think very seriously before scribbling some illegible data on an inadequate piece of paper after the patient has left the operating room.

In summary, I approached this book without much enthusiasm but found myself enjoying it enormously and I am sure I gained a great deal from it.

J.C. Stoddart

New Neuromuscular Blocking Agents. Handbook of Experimental Pharmacology, Volume 79

Edited by D.A. Kharkevich. Pp. 741. Springer-Verlag, 1986. DM 780.–.

This is an extra-ordinary book in many ways. To begin with, just over half of the 36 contributions are from Eastern Europe and its Editor is the Professor of Pharmacology at the First Medical Institute in Moscow (Dimitry Kharkevich). Then it costs more—about £270 at current exchange rates—than I paid for my first car (a cantankerous, blue, 1955 Ford Prefect). And its content is unique with (hundreds of) pages devoted to compounds which are quite unfamiliar to most of us in the West. Although of some interest (and occasionally intriguing) much of this information is of little practical value, even to those of us active in this area.

After reading the book, one's lasting impression is of a Central Europe carpeted with plants which contain alkaloids that either inhibit or restore neuromuscular transmission. It is Volume 79 of a series entitled *Handbook of Experimental Pharmacology* published by the German house of Springer-Verlag. Previous volumes have dealt with: Arthropod Venoms (Volume 48), Snake Venoms (Volume 52) and Pharmacology of Intestinal Permeation (Volume 70). It is building into a unique series.

After a couple of introductory chapters, which include a useful review of acetylcholine receptors, the remaining 31 chapters are divided into five sections: Pharmacodynamics and pharmacokinetics of neuromuscular blocking agents; On the relationship between the chemical structure and the neuromuscular blocking activity (*sic*); Preclinical pharmacology of new neuromuscular blocking drugs; Clinical pharmacology of new neuromuscular blocking drugs; Antagonists of neuromuscular blocking agents. These subdivisions are rather arbitrary (and unhelpful) and there is a lot of overlap between the sections. For instance, there are three similar accounts of derivatives of truxillic acid (from a Central Asian plant *Thesium minkwitzianum*). This is just one example amongst many which could be cited.

The Editor justifies the inclusion of outdated information from Eastern authors on the grounds that it has previously been inaccessible to most Western readers. This is a valid point of view but it must be stated that there is rather too much of this sort of writing: 'At the same time, taking into consideration the fact that the skin remains dry and warm with certain clinical signs of adequate circulation, one can assume that the hypotensive effect and ganglion-blocking properties of anatruxanium are closely connected.' However, there are quite a few good reviews, some of which could not be found easily from other sources. These include chapters on the anticholinesterase, galanthamine (by Paskov of Rumania) and on the long-acting steroid relaxant, pipecuronium (by three authors from Budapest). The accounts of atracurium (by R. Hughes) and vecuronium (by R.D. Miller) are workmanlike, but a bit dated now—the latest references that they cite are from 1983 and 1982, respectively. However, with a multi-author book, especially of this nature, some delay in collecting and publishing material is inevitable and the Editor has undertaken a gargantuan task for which he deserves respect.

In conclusion, this outrageously expensive book contains a vast amount of material not readily available from other sources. A significant proportion of this material is of questionable value, except to students of the recent history of muscle relaxants in Eastern Europe. It is not for the shelves of the average departmental library, although active researchers in the field might find it useful to have access to a copy.

R.M. JONES

Primary Anaesthesia

Edited by M.H. KING. Oxford University Press, 1986. Indexed, illustrated. Pp. 169. £7.95.

There has long been a requirement for a comprehensive manual of modern anaesthesia for medical staff in developing countries. This large-format volume goes a considerable way towards fulfilling that need. Produced under the sponsorship of the German Agency for Technical Cooperation (GTZ) and the Kenyan Ministry of Health, it draws on the knowledge of many anaesthetists (and surgeons) suitably experienced in the appropriate techniques.

Punctuated with illuminating case histories and exhortations, the correct emphasis on safety and simplicity is maintained throughout. The advantages of drawover systems and clinical monitoring techniques over the high-tech approach are expounded and emergency drills described in each section. The basic sciences are well covered and equipment is described thoroughly. There is a useful section on purchasing, which is wisely confined to essentials, and instructions on the manufacture of intravenous fluids.

Deficiencies are few: the section on paediatric anaesthesia is rather brief, and strangely, considering the number of British contributors, there is no mention of the Triservice Apparatus. The otherwise excellent section on epidural anaesthesia contains the rather unusual advice that an epidural catheter is optional for Caesarean section.

The whole book is laid out in a logical and attractive manner with many illustrations. The relationship between experience and suitability of techniques is constantly reinforced, and the extent of knowledge acquired can be assessed by the multiple-choice questions included.

Most anaesthetic departments in this country employ doctors from developing countries, but give little thought to the applicability of a British training to their future careers. This excellent and sensibly priced book would help redress the balance and deserves a prominent place in every department's library.

N. PUTTICK

Regional Anaesthesia, Techniques and Clinical Applications

H. CARRON, G.A. KORBON AND J.C. ROWLINGSON. Pp. 198. Grune and Stratton, Inc., 1984. £33.00.

This book was published in 1984 and it is the authors' stated intention to provide a text that will show pertinent anatomical considerations, surface landmarks and techniques for performing regional anaesthetic procedures. They freely admit that they employ a cookbook approach, but argue that reproducible routines lead to successful regional anaesthesia, and in this opinion your reviewer concurs. However, to ensure success, accurate and reliable information must be provided and in certain sections of this book it is lacking.

This reviewer considers that any book that is aimed at the trainee anaesthetist (and this book is no exception, being dedicated to them in particular), must contain considerable details of the drugs used, and how to use them, as well as exhaustive discussion of the problems that may be encountered and how to deal with them. It would seem reasonable to give plenty of detail on common and useful blocks, and expend less space on those more likely to be used by the experienced anaesthetist. Obviously this is a matter of opinion but, as an example, less than five pages of text and ten references (the latest of which is 1977) in the introductory chapter on pharmacology and choice of local anaesthetic agent, hardly seems adequate to provide the trainee anaesthetist with a solid grounding for clinical practice or the experienced anaesthetist with any useful information.

The main body of the text is divided into sections on plexus blocks, neural axis blocks, peripheral blocks, pain blocks, enhancing regional procedures and complications. It is obvious from some sections that the authors use the blocks they describe on a day-to-day basis, but in others the practical detail leaves much to be desired. The opening section is an example of this, with irritating omissions from the descriptions of the various techniques of brachial plexus blockade that would undoubtedly affect success. When the interscalene and supraclavicular approaches to the brachial plexus are described, the authors ignore Winnie's concept of a sheath around the brachial plexus and suggest the injection of local anaesthetic solution on the elicitation of a paraesthesia, or on contact with a specific bony landmark—the transverse process of C_6 and the first rib, respectively—both of which may be some way from the sheath which contains the nerves. Little guidance is offered on how to maximise the likelihood of obtaining a paraesthesia anatomically and, thus, the chances of failure must be increased. The use of the immobile needle is not mentioned until much later in the book, in the chapter on needles. There is no mention of aspiration before and during injection, and no help is given on how to detect intraneural injection until the penultimate chapter.

There are many other examples that reflect differences between current British practice and American practice of a few years ago. Three agents are mentioned for spinal use: procaine, tetracaine and lignocaine. Only the latter is available in Britain and only on a limited basis. The authors recommend bupivacaine for intravenous regional anaesthesia and this section contains the only reference to prilocaine in the text! In the special procedures section only the most rudimentary indications are given for each procedure; surely it important to know why a procedure is being perf and what are the expected benefits, as it is to how to do it.

The techniques described undoubtedly refle experience of the authors and this must be resp The differences and omissions may also reflect ch in practice since the book was written. There are sections on pain and neurolytic blocks, althou views are a little elderly. The section on enh regional techniques is well done, but is too bri would have been better as one of the first section giving the beginner every aid to success in hi attempt.

In summary, this is a disappointing book b may be because better texts are now available a book suffers by comparison. There is much wort information but one is left with the impressio important information has been sacrificed for th of brevity. The illustrations are small but usuall ful and there are 52 eclectic suggestions for f reading.

J.E. CHA

Anaesthesia, 1987, Volume 42, pages 335–340

Anaesthetic literature

This section of *Anaesthetic literature* contains references taken from *Current Contents—Life Sciences* for October 1986. The journals which may be consulted when *Anaesthetic literature* is compiled are those listed in *Current Contents—Sciences*, published by the Institute of Scientific Information, Philadelphia, Pennsylvania, USA.

Abdominal surgery

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Comparison of different methods of postoperative analgesia after thoracotomy. ASANTILA, R., ROSENBERG, P.H., SCHEININ, B. *Acta Anaesthesiologica Scandinavica* 1986; **30:** 421.

Other

Treatment and medication

Emergency management of accidental hypothermia: a review. MACLEAN, D. *Journal of the Royal Society of Medicine* 1986; **79:** 528.

Anaesthesia, 1987, Volume 42, page 341

Errata

Anaesthesia, 1986, Volume 41, pages 1026–1028

Anaesthesia and the subclavian steal syndrome

R. J. THOMPSON

The second sentence of paragraph two in the first case history (*Case 1*, p. 1026), should read: ... anaesthesia was maintained with nitrous oxide 6 litres/minute plus oxygen 3 litres/minute and trichloroethylene.

Anaesthesia, 1986, Volume 41, pages 995–1000

Comparison of induction characteristics of four intravenous anaesthetic agents

J. S. C. McCOLLUM AND J. W. DUNDEE

The legend to Figure 2 and the initial part of that to Figure 1 were inadvertently transposed. The correct figure captions should have appeared as:

Fig. 1. Induction grades in patients in whom an adequate depth of anaesthesia was induced. □, Uncomplicated; ▦, mild upset; ■, moderate upset; ■, completely unsatisfactory.

Fig. 2. Percent systolic blood pressure change from baseline in individual patients, with mean.

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The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36:** 119–25.

Anonymous. Epidemiology for primary health care. *International Journal of Epidemiology* 1976; **5:** 224–5.

BOOKS AND OTHER MONOGRAPHS

Personal author(s)

OSLER AG. *Complement: mechanisms and functions.* New York: Prentice-Hall, 1976.

Corporate authors

American Medical Association Department of Drugs. AMA drug evaluations, 3rd edn. New York: Publishing Sciences Group, 1977.

Editor, compiler, chairman as author

RHODES AJ, VAN ROOYEN CE, *comps. Textbook of virology: for students and practitioners of medicine and other health sciences,* 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

Chapter in book

WEINSTEIN L, SWARTZ MN. Pathogenic properties of invading micro-organisms. In: SODEMAN WA Jr, SODEMAN WA, eds. *Pathologic physiology: mechanisms of disease.* Philadelphia: W.B. Saunders, 1974: 457–72.

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National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968—June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series* **10:** Data from the National Health Survey, No. 69) [DHEW publication No. (HSM) 72–1036].

OTHER ARTICLES

Newspaper article

SHAFFER RA. Advances in chemistry are starting to unlock mysteries of the brain: discoveries could help to cure alcoholism and insomnia, explain mental illness. How the messengers work. *Wall Street Journal* 1977 Aug 12: 1(col 1), 10(col 1).

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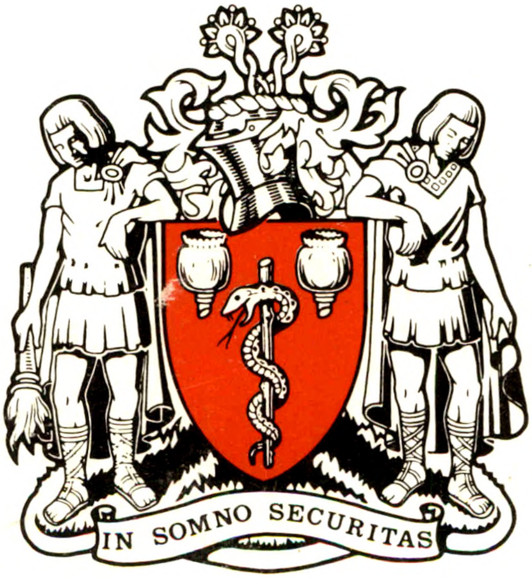

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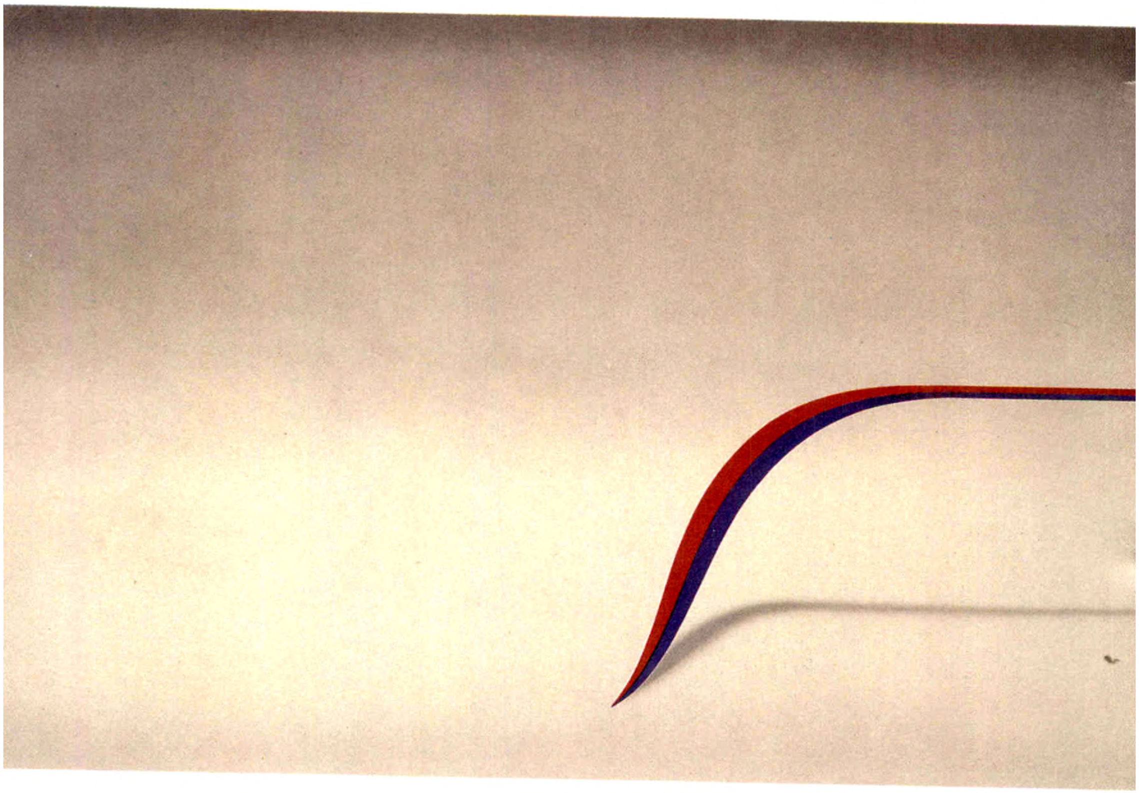

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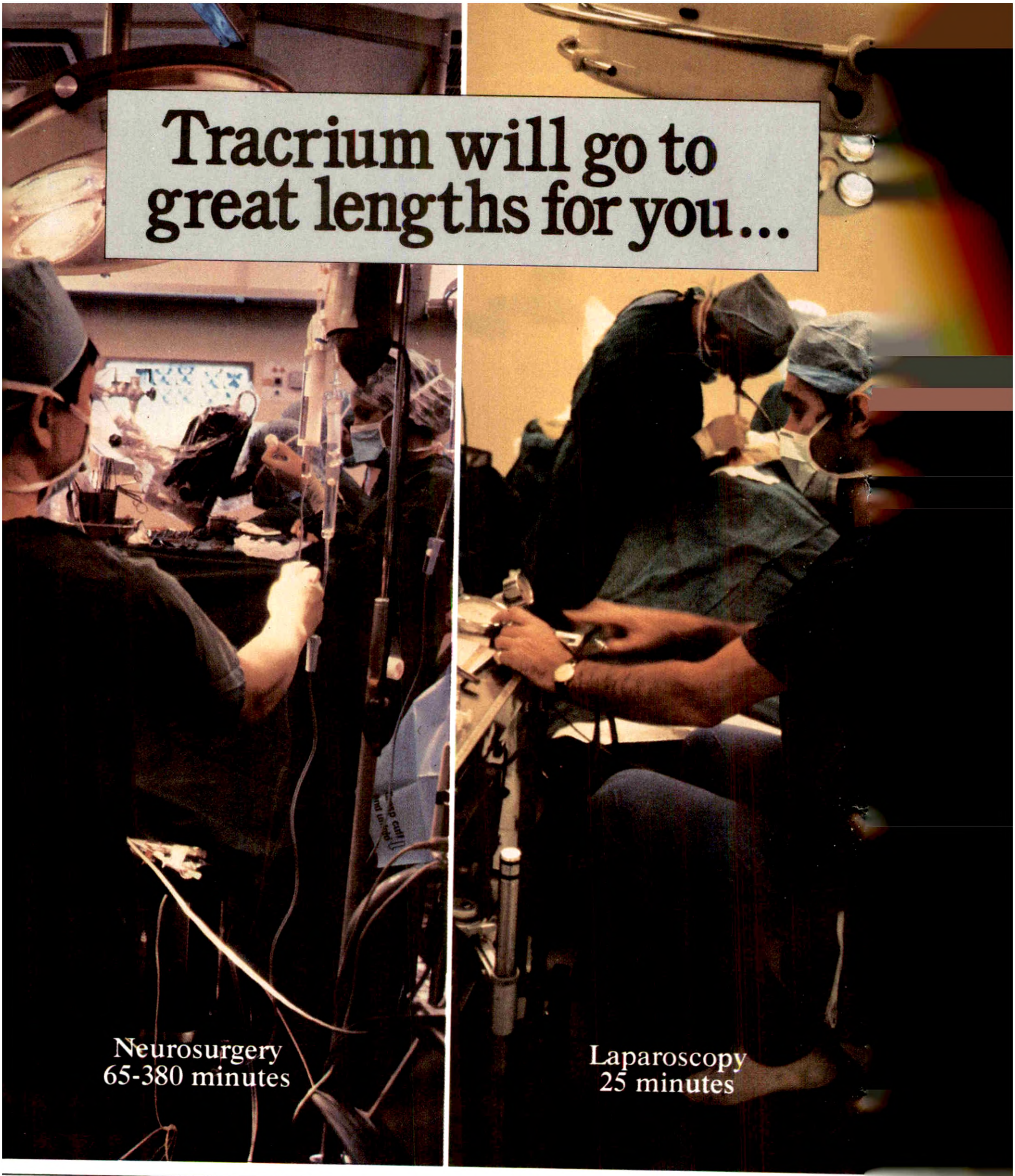

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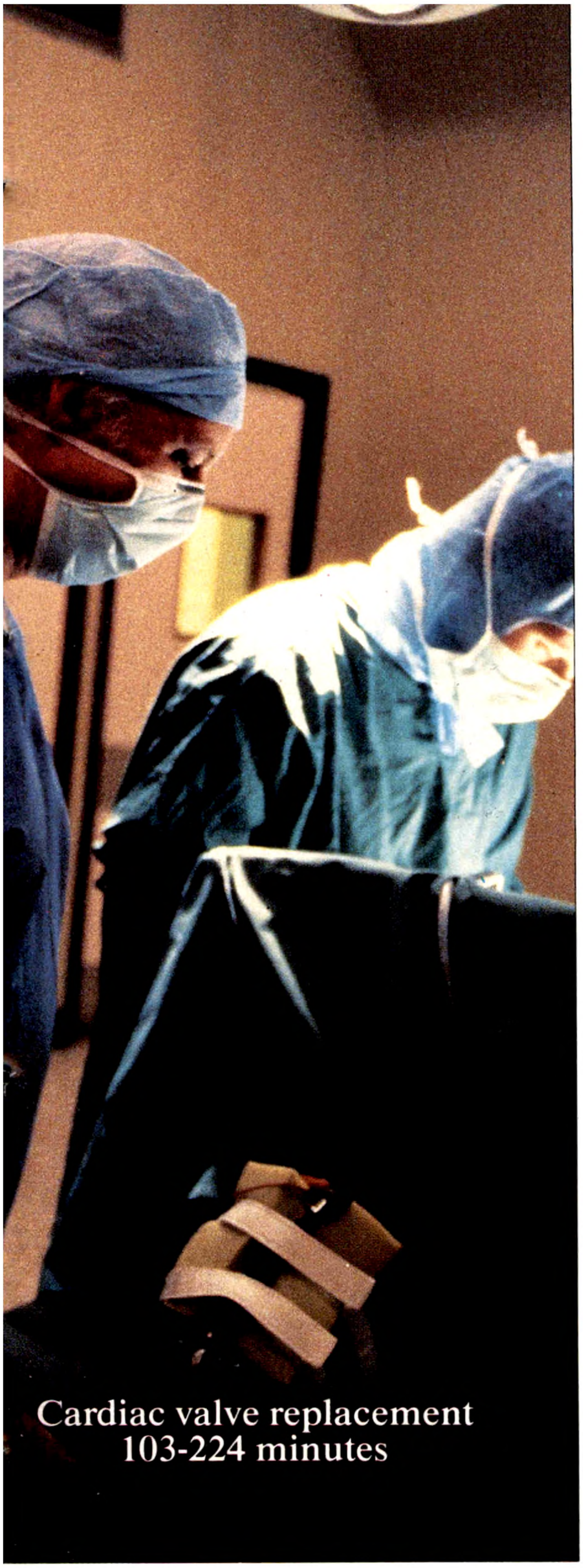

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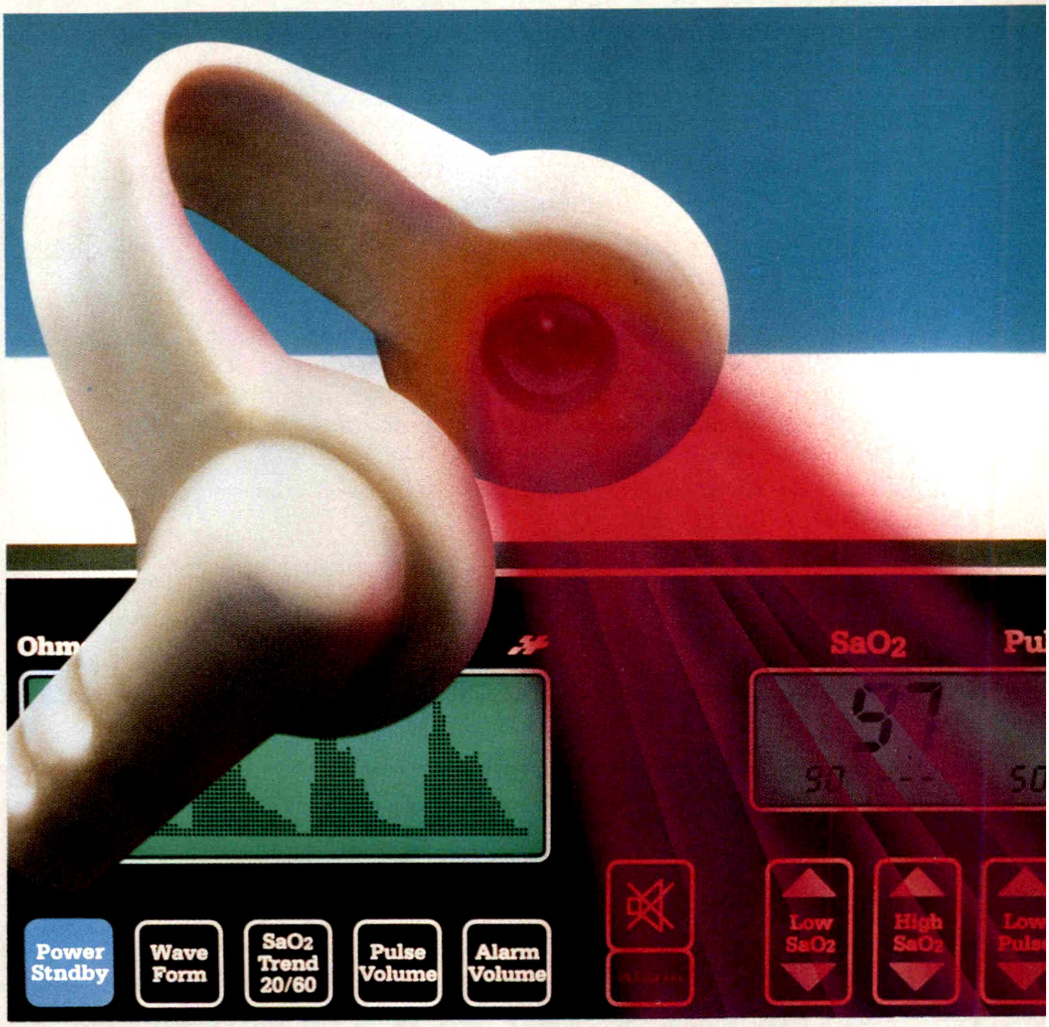

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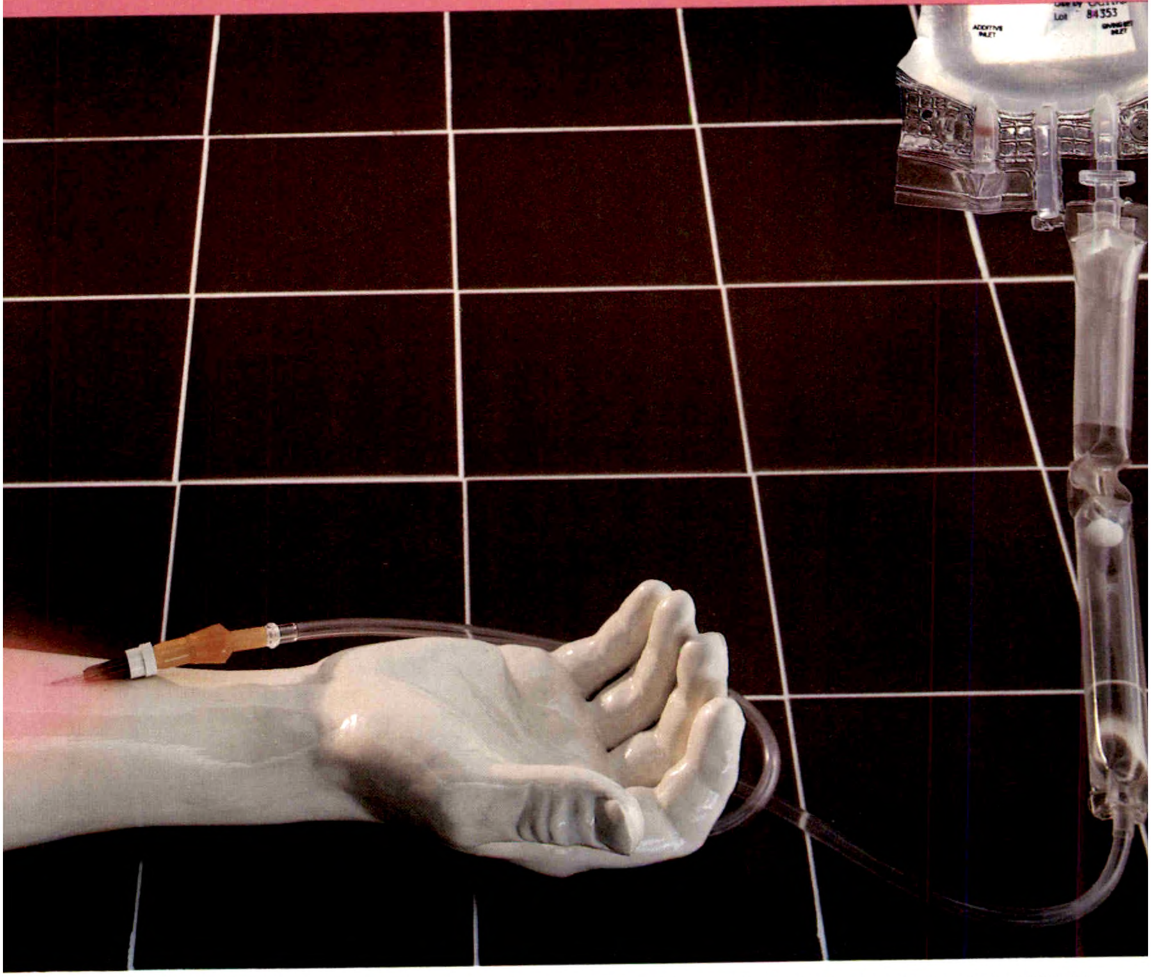

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Anaesthesia, 1987, Volume 42, pages 343–345

Editorial

Postoperative analgesia for neonates?*

Recent work by Anand and Aynsley-Green[1] which demonstrated a significant neonatal endocrine response to stress, has focussed our attention, yet again, on the vexed question of whether or not neonates feel pain. It seems that, at the very least, preterm neonates subjected to surgical stress mount a substantial and prolonged catabolic reaction in the postoperative period, which is more marked if little or no anaesthesia or analgesia is given. This must give us food for thought.

In a recent survey of British paediatric anaesthetists, it appeared that 48% of respondents never provided postoperative analgesia of any kind to neonates during or after major surgery.[2] Thus there are obviously some anaesthetists who feel that the newborn human is fundamentally different from the rest of the species with regard to the perception of pain, but is this premise a result of rational thinking based upon observed facts, or upon unproven hypotheses? What evidence have we that the neonate does, or does not, feel pain?

Certainly the neonate has the anatomical equipment necessary to feel pain. He has a cerebral cortex, thalamus, spinal cord, peripheral nerves and receptors and has developing neuronal connexions to link them. The question remains: do these neuronal connexions function in a way that is analogous to that of the adult?

There have been many studies of the normal response to painful stimuli in the newborn, albeit limited by ethical and practical considerations. Reactions to repeated pinprick in the lower limbs include an increase in heart rate, movement of upper and lower limbs, facial grimacing and crying. Changes in skin conductivity and transcutaneous $P\text{O}_2$ have also been noted. Facial expressions associated with various emotional states, including pain, have been reliably differentiated by experienced observers. Attempts made to classify cries according to their acoustical properties, using the technique of sound spectrography, offered the prospect of a highly sensitive and quantitative method for the study of crying. However, it appears that cries are not acoustically unique, although they vary in intensity according to the amount of discomfort experienced by the neonate.[3] Behavioural studies on neonates who have been circumcised have shown that changes in behaviour may persist for more than 22 hours postoperatively in babies who have received no analgesia.[4]

So what can we conclude from these studies? It has been argued that many of these responses represent simply reflexes to noxious stimulation and do not necessarily imply distress. However, behavioural changes and crying are responses that do involve higher nervous system function. Nevertheless, because a functioning cerebral cortex is necessary for sensation and perception, direct evidence that cortical activity does occur in the neonate is needed.

Cortical activity in response to visual stimuli has been demonstrated in babies from the 25th week post-conception. Similarly, it has been shown that other stimuli, including auditory, olfactory and tactile, result in cortical electroencephalographic activity in preterm neonates.[5] Indirect evidence of neonatal cortical activity also comes from behavioural studies. Neonates track objects with their eyes and can distinguish their mother's face from that of another female similarly presented. Neonates can distinguish voices over other sounds, and female over male voices. They can also imitate tongue protrusion, which suggests that they perceive the equivalence of another person's face to their own face.[6] Additional evidence of motor cortical activity comes from experiments on newborns with exencephaly, where isolated movements of digits have resulted from direct electrical stimulation of cortical tissues. Motor cortical activity is also suggested in neonates with convulsions, as cortical convulsive potentials occur synchronously with peripheral massive muscle contractions.

There can be no doubt, therefore, that various cortical areas of the brain are active in the neonatal

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* This Editorial is based on discussions during a Seminar on this subject held at 9 Bedford Square in October 1986.

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period, and the conclusion we must therefore consider is that the neonate could well have the ability to perceive pain at a cortical level. It has been argued that although babies may feel pain, this does not matter since memory of the sensation is not retained and that memory is necessary for the development of adult-like pain perception. Conversely, other workers have speculated that psychological sequelae may follow severe pain experienced in the neonatal period. Both proposals remain conjectural at the present time, although some long-term behavioural consequences of unanaesthetised newborn circumcision have been suggested by studies of infant gender differentiation.[7]

How may we tell if a neonate is in pain? Signs of distress such as crying, facial grimacing and tachycardia, when taken in the context of likely or actual tissue damage, are helpful. Other useful behavioural indicators include restlessness, being slow to settle after stimulation, and unusually long periods of wakefulness.

The provision of effective pain relief for the spontaneously breathing neonate can be dangerous and this is, no doubt, why some anaesthetists refrain from prescribing any analgesia at all. The increasing use of local anaesthesia in paediatric practice in general does not seem to have reached the neonate to a similar extent, partly because of technical difficulties and partly because of the reduced plasma protein binding and prolonged elimination of bupivacaine in this age group. However, these problems are not insuperable and the writer advocates the use of local blockade whenever possible. Paracetamol is one analgesic that is safe to use in the non-jaundiced neonate, although its efficacy in the immediate postoperative period may be open to question. The administration of an opioid is more controversial because of the marked variation in the response of the neonate to this group of drugs. However, for those babies who require controlled ventilation of the lungs after major surgery, an opioid infusion can be considered, since then respiratory depression is not a problem and indeed could be advantageous.

In conclusion, if the assumption that these young babies experience pain is accepted, then we should do all we can to decrease needless human suffering. Moreover, by providing postoperative analgesia, we may be able to reduce the endocrine–metabolic stress responses, which are not always beneficial. At the very least, we need to increase research into new, safe methods of effective analgesia for this age group.

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Editorial notices

The Editor and the Assistant Editors are grateful to many informal advisers and to the following who have provided formal assessments of manuscripts over the past year.

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Submission of manuscripts

Manuscripts must be submitted in accordance with the internationally recognized *Uniform requirements for manuscripts submitted to biomedical journals* (*British Medical Journal* 1979; **1:** 532–5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

Anaesthesia News

A new service for members of the Association of Anaesthetists of Gt Britain and Ireland is announced. This news sheet will be distributed free with members' copies each quarter. It replaces the News and Notices section of the Journal, with the exception that Obituarial Notices and the International Calendar will continue to be published quarterly. Publication of the Annual Report of Council and Linkman Conferences will also continue.

Instructions for submissions to *Anaesthesia News* are included in the first number, which appears with this issue of *Anaesthesia*.

Anaesthesia, 1987, Volume 42, pages 346–351

Pre-oxygenation and the parturient patient

G. N. RUSSELL, C. L. SMITH, S. L. SNOWDON AND T. H. L. BRYSON

Summary

The effects of pre-oxygenation were studied by continuous expired oxygen analysis in twenty pregnant patients and ten nonpregnant controls. The Magill system and a demand valve breathing system were both studied with tidal and vital capacity breathing. Rapid pre-oxygenation was possible with the demand valve system but the technique was associated with air leaks around the mask. Vital capacity breathing with the Magill system was inefficient in practice because of rebreathing and air leaks, and the recently proposed four vital capacity breath technique provided suboptimal alveolar oxygenation in pregnant subjects. Oxygenation in pregnant subjects was significantly faster than in nonpregnant controls and was achieved after just 2 minutes of tidal breathing from a standard Magill system with a prefilled reservoir. Care was necessary to ensure a gas-tight seal around the facemask.

Key words

Oxygen; breathing systems.
Anaesthesia; obstetric.

Most of the published work on the effects of pre-oxygenation has concerned the nonpregnant general surgical patient.[1,2] We feel, however, that the pregnant patient deserves special attention for three reasons. First, pre-oxygenation has become a standard feature in the rapid sequence induction of general anaesthesia for Caesarean section. Second, in the acute obstetric emergency, failure to achieve maximum pre-oxygenation can have grave consequences. Third, term pregnancy is associated with changes in lung volumes and function. The mean tidal volume is increased by about 40% over values found in the nonpregnant,[3] due probably to progesterone.[4] Upward displacement of the diaphragm by the gravid uterus causes a reduction in functional residual capacity (FRC) to 9.5–25% below the values obtained in nonpregnant subjects.[3,5,6] The reduction in FRC associated with relative alveolar hyperventilation should facilitate rapid pre-oxygenation prior to Caesarean section.

Two breathing systems were investigated. One was a system that incorporates both demand and non-rebreathing valves[7] similar to the standard Entonox apparatus. This permits a peak inspiratory flow of 500 litres/minute without rebreathing of expired gas. As such, it should theoretically be the ideal system for pre-oxygenation. The Magill system was also investigated because it is probably the breathing system most widely used for pre-oxygenation in the United Kingdom.

This study was designed to assess the opti-

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mum duration and technique for pre-oxygenation prior to Caesarean section with these two breathing systems, and to compare the rate of oxygenation with nonpregnant controls.

Patients and methods

The study was approved by the hospital ethical committee. Twenty pregnant volunteers of 38–41 weeks gestation, as estimated by ultrasound scan, were studied. They were all singleton pregnancies and not in labour; pregnancy was uncomplicated and they had been admitted to hospital for induction. In addition, ten midwives volunteered to act as nonpregnant controls. None of the subjects had significant cardiorespiratory disease. The age, height and nonpregnant weight of all subjects were noted.

Oxygenation was assessed by continuous end expired oxygen analysis using a Medishield MS2 quadrupole mass spectrometer. The instrument was allowed to stabilise for at least 3 hours prior to calibration with oxygen and room air. Calibration was rechecked between each set of breathing measurements. Gas was sampled at 30 ml/minute via a port in the anaesthetic facemask, next to the lips. The electronic read-out from the mass spectrometer was displayed on a chart recorder to provide a permanent record of the wash-in curve obtained from each breathing technique.

During oxygenation with the Magill system, a fresh oxygen flow of 8 litres/minute was supplied from a calibrated flowmeter (this is the maximum flow which can be measured by the rotameter on many anaesthetic machines currently in use). Both tidal and vital capacity breathing were studied with this system. Tidal breathing was obtained by asking subjects to breathe normally from the system, with the reservoir initially either empty or filled with oxygen prior to application of the facemask. Vital capacity breathing was studied with the standard 2-litre reservoir bag and also with a 4-litre reservoir bag. Subjects were instructed to take slow, maximal inspirations and full expirations with the reservoir bag prefilled with oxygen. The subjects held the facemask unaided after a short explanation of the technique required, and no attempt was made to control the exact respiratory rate.

The system with demand and non-rebreathing valves was coupled to an oxygen cylinder. Large minute volumes should be practical with this system and so, by instructing the subjects to take rapid, deep breaths, it was possible to study rapid vital capacity breathing.

Thus, a total of five breathing measurements were made on each subject, each acting as her own control. Four measurements involved the Magill system—tidal with reservoir bag (2-litre) prefilled, tidal with reservoir bag initially empty, and slow vital capacity breathing with 2-litre and 4-litre reservoir bags. The fifth measurement was rapid vital capacity breathing from the demand valve system. At least 5 minutes breathing room air was allowed between the techniques, and the return to baseline end expired oxygen concentration was confirmed using the mass spectrometer before starting the next measurement. All subjects were studied in the supine left tilted position, and breathing techniques investigated in random order.

Breath volume during tidal breathing was measured with a calibrated electronic Wright's spirometer (British Oxygen Co. Ltd) at the angle piece of the breathing system. The tidal volume was displayed concurrently with the wash-in curve on the chart recorder.

The wash-in curves obtained were examined by an analyst who was unaware of whether the subject was pregnant or not. The endpoint for optimum pre-oxygenation was taken to be the onset of the plateau phase of the exponential wash-in curve, and was defined as the breath after which the end expired oxygen concentration increased by less than 1% over the subsequent 30 seconds. The time and number of breaths to this point were noted. The time to achieve an end expired oxygen concentration of 85% was noted to aid comparison, particularly during slow vital capacity breathing. In addition, the end expired oxygen concentration achieved after four vital capacity breaths was noted. Student's *t*-test was used for statistical comparisons, with $p < 0.05$ taken to be significant.

Results

There were no significant differences between the two study groups in age, height or nonpregnant weight (Table 1). The two groups and individual breathing techniques are compared in Table 2. Examples of the wash-in curves obtained from pregnant patients using the Magill

Table 1. Subject data. Values are mean (SD).

	Pregnant (n = 20)	Nonpregnant (n = 10)
Age, years	27 (6)	25.5 (2)
Height, cm	159 (5)	164 (8)
Nonpregnant weight, kg	66 (8)	61 (5)
Tidal volume, litres	0.77 (0.20)	0.56 (0.12)
Respiratory rate	14 (3.3)	15 (4)

system are shown in Figs. 1–4. The pregnant subjects had significantly larger ($p < 0.05$) tidal volumes than the nonpregnant controls (Table 1).

Optimum oxygenation was achieved significantly faster by the pregnant group ($p < 0.05$) with all breathing techniques on the Magill system. Pregnant subjects reached the plateau phase of the wash-in curve after a mean of 90 seconds of tidal breathing from the Magill system if the reservoir bag was prefilled with oxygen. Failure to prefill the reservoir, significantly reduced the rate of oxygen wash in ($p < 0.05$).

The rate of oxygen wash in during slow vital capacity breathing on the Magill system was not significantly faster than during normal tidal breathing from a prefilled reservoir. The mean end expired oxygen concentration of 79% achieved in pregnant patients after four vital capacity breaths (Table 3), was significantly lower ($p < 0.001$) than could be achieved if the patients were optimally oxygenated to the plateau phase.

Table 3. Fractional end expired oxygen concentration in 20 pregnant patients achieved after four vital capacity breaths compared with oxygenation to plateau phase.

	Mean (SD) $F\text{E}'\text{O}_2$ after four vital capacity breaths	Mean (SD) $F\text{E}'\text{O}_2$ at onset of plateau phase
2-litre reservoir, full vital capacity breaths	0.79 (0.035)*	0.90 (0.01)
4-litre reservoir, full vital capacity breaths	0.79 (0.04)*	0.89 (0.015)

*$p < 0.001$. Comparison with values obtained at plateau phase.

Hyperventilation with the demand valve system allowed very rapid pre-oxygenation in both pregnant and nonpregnant patients. The

Table 2. Results from the wash-in curves for pregnant and nonpregnant groups. The five breathing techniques are compared. Values are mean (SD).

	Pregnant (n = 20)					Nonpregnant (n = 10)				
	Tidal		Vital capacity			Tidal		Vital capacity		
	Reservoir full	Reservoir empty	2-Litre reservoir	4-Litre reservoir	Demand valve	Reservoir full	Reservoir empty	2-Litre reservoir	4-Litre reservoir	Demand valve
Time in seconds to plateau phase	90 (17)	106 (14)*†	92 (14)	96 (18)	56 (8)**†	124 (21)	140 (28)	116 (15)	112 (18)	53 (6)
Number of breaths to plateau phase	21 (5)	25 (5)	9 (1.5)	9 (2)	15 (5)	31 (12)	35 (12)	11 (3)	10 (3)	25 (5)
Time in seconds to $F\text{E}'\text{O}_2$ 0.85	59 (14)	69 (15)	61 (16)	59 (14)	30 (14)	80 (23)	90 (22)	69 (21)	70 (21)	25 (9)

*$p < 0.05$. **$p < 0.001$.

† Comparison with tidal breathing from a prefilled reservoir bag. For other comparisons between groups, see text. $F\text{E}'\text{O}_2$, fractional concentration of expired oxygen measured at the facemask.

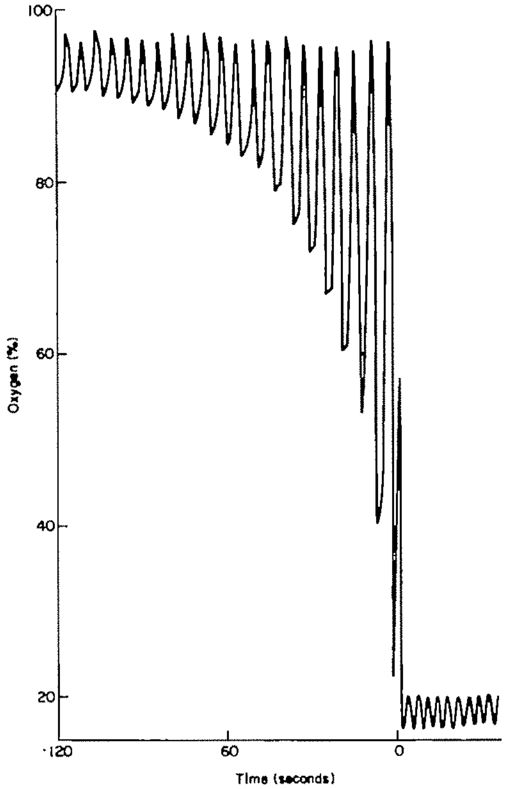

Fig. 1. The wash-in curve obtained during tidal breathing from a prefilled reservoir on the standard Magill system. The peaks represent inspired and the troughs end expired oxygen concentration. High inspired oxygen concentrations are rapidly achieved with this technique.

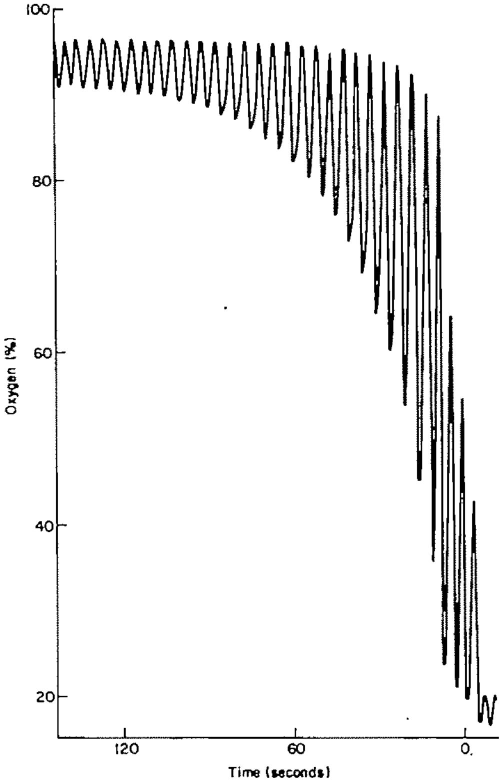

Fig. 2. The wash-in curve during tidal breathing from the Magill system with the reservoir initially empty. Rebreathing of expired gases slows the rise in inspired and alveolar oxygen concentrations.

plateau phase in the former group was achieved after a mean of 56 seconds, a significant improvement on any of the techniques that used the Magill system ($p < 0.001$). No difference between the two study groups was found in the rate of pre-oxygenation with the demand valve system.

Discussion

The aim of pre-oxygenation is to increase the oxygen stores of the body in order to increase the duration of apnoea that will be possible without the onset of hypoxia. The FRC is the main oxygen store of the body, and continuous expired oxygen analysis allows a wash-in curve for the lungs to be constructed and analysed.

The oxygen wash-in curve for the normal lung is exponential[8] with an initial fast phase followed by a plateau (Fig. 1). Once the plateau phase has been reached, pre-oxygenation can be said to be optimum. Continued oxygenation will not then achieve a clinically significant improvement in alveolar oxygenation. The end expired oxygen concentration may rise to over 90% in pregnant subjects due to efficient wash out of the FRC and to the reduction in alveolar carbon dioxide concentration associated with pregnancy.[9]

The time constant for the wash-in curve is a function of the FRC divided by the alveolar ventilation.[8] As a result of the physiological changes associated with normal breathing at term pregnancy, namely a decrease in FRC and an increase in alveolar ventilation, the time constant for the lung is shortened—therefore, oxygenation should be more rapid in this group. Our results support this hypothesis in patients

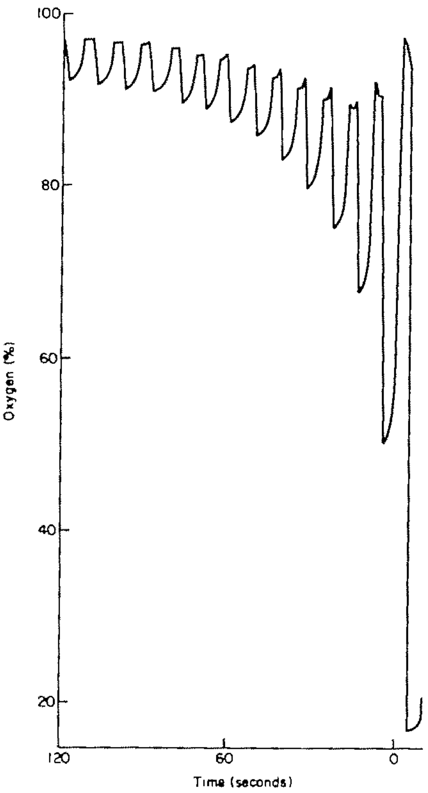

Fig. 3. Slow vital capacity breathing with the 2-litre reservoir bag. Fewer breaths are required to achieve optimum oxygenation but the rate of oxygenation is not increased.

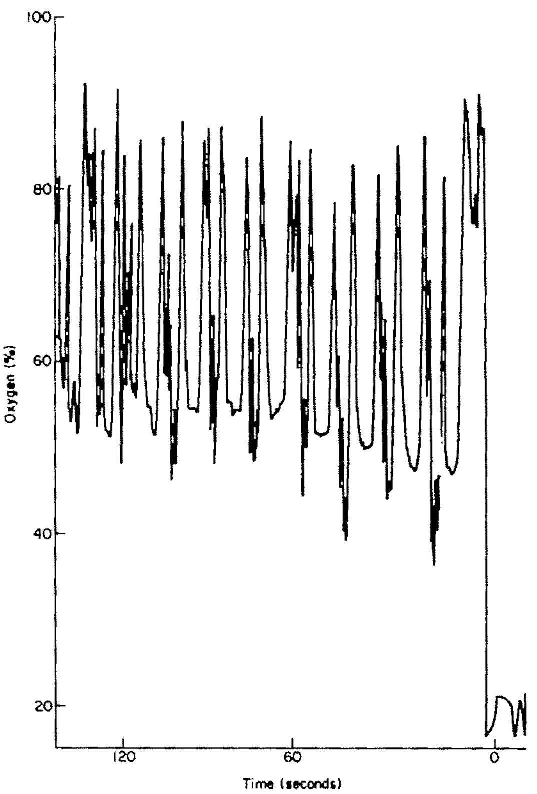

Fig. 4. The effect of an air leak during vital capacity breathing with the Magill system. Entrained room air reduces the inspired oxygen concentration and results in unpredictable alveolar levels.

who breathed normally through the Magill system (Table 2), and the values obtained in nonpregnant subjects are similar to those found by others who used expired gas analysis.

No significant difference in the time to optimum oxygenation was observed between pregnant and nonpregnant subjects when they hyperventilated through the demand valve system. Our nonpregnant controls, however, had a significantly faster respiratory rate ($p < 0.05$) and the consequent increase in alveolar ventilation probably overshadowed any difference in static lung volumes between the two groups.

By reducing the inspired oxygen concentration, rebreathing nitrogen-rich exhaled gas will slow the rate of oxygenation. During tidal breathing the use of a prefilled reservoir bag (Fig. 1) prevented the initial period of rebreathing seen when a virtually empty reservoir is used (Fig. 2) and allowed a significantly improved rate of oxygenation (Table 2). During vital capacity breathing the first breath achieves a high inspired oxygen concentration; rebreathing is then evident by the reduced inspired oxygen concentration of the second and subsequent breaths (Fig. 3). This would be worse at higher minute volumes[10] and could be lessened by the use of an oxygen flush facility to increase the fresh gas supply. However, we found the use of the oxygen flush both inconvenient and uncomfortable for the patient, and abandoned its use following our pilot study.

At first, rapid vital capacity breathing was tried on the Magill system so as to increase the rate of oxygenation, but large air leaks were observed around the facemask (Fig. 4). Room air was entrained because of the high inspiratory flow rate and the negative pressure created within the system, especially if the reservoir bag collapsed. The inspired oxygen concentration was hence variable and decreased, and limited the rise in alveolar concentration. In contrast, slow vital capacity breathing (Fig. 3) reduced the incidence of air leaks, although we did not observe a significant reduction in the time taken to reach the plateau phase compared with tidal breathing from a prefilled reservoir bag. Similarly, the use of a 4-litre reservoir bag did not improve the rate of oxygenation and was cumbersome in clinical practice.

Gold *et al.*[11] advocated the use of just four vital capacity breaths for pre-oxygenation of the nonpregnant patient. In our study of pregnant patients, however, it proved to be the least effective technique, and resulted in the lowest end expired oxygen concentration (Table 3). The reduction in FRC at term pregnancy implies a reduced lung oxygen reservoir which, in association with an increased basal oxygen consumption,[12] makes this group particularly prone to hypoxia. We would not, therefore, recommend the routine use of the four vital capacity breath technique for the pregnant patient in whom pre-oxygenation should be optimum; it may be applicable to the acute clinical emergency if the saving in time is judged to outweigh the consequences of the smaller oxygen reservoir achieved.

Vital capacity hyperventilation with a demand valve system is one of the most rapid techniques of pre-oxygenation available in clinical practice but, in common with all vital capacity modes, it suffers from a high incidence of air leaks and requires special care to ensure a gas-tight seal around the facemask. The requirement for an additional breathing system in the operating theatre may be unpopular.

In summary, slow vital capacity breathing has no overall advantages as a pre-oxygenation technique when compared with tidal breathing from a prefilled reservoir. The use of the four vital capacity breath technique is not supported by this study. Ninety-five percent of pregnant patients will be optimally pre-oxygenated with 2 minutes of normal tidal breathing from the standard Magill system with a prefilled reservoir and 8 litres/minute fresh gas flow, if care is taken to avoid air leaks around the facemask.

Acknowledgments

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Anaesthesia, 1987, Volume 42, pages 352–355

Antacid therapy for emergency Caesarean section

J. THORBURN AND D. D. MOIR

Summary

One hundred patients undergoing emergency Caesarean section were treated with cimetidine 200 mg intramuscularly when the decision was made to deliver the patient by Caesarean section and, preceding general anaesthesia, 30 ml of 0.3 M sodium citrate was administered orally. No routine antacid therapy was employed during labour. No patient had a gastric aspirate pH of less than 2.7, and only one of less than 3. The regimen is simple and effective, and treats only those patients at risk of acid aspiration.

Key words

Anaesthesia; obstetric.
Pharmacology; cimetidine, sodium citrate.

Despite the recognition of the effects of aspiration of acid gastric contents associated with obstetric anaesthesia by Mendelson,[1] and the almost universal adoption in the UK of the prophylactic use of antacids orginally advocated by Taylor and Pryse-Davies,[2] it is disappointing to record continuing failure to reduce significantly obstetric mortality from aspiration of gastric contents.[3] Indeed, in that report, urgent review of antacid therapy was advised.[3] The majority of studies of the efficacy of antacid therapy have testified to the superiority of magnesium trisilicate, which is particulate and the most widely used antacid in the UK. However, Gibbs *et al.*[4] have demonstrated that adverse pulmonary effects follow the aspiration of particulate antacids. Microscopic examination of the pulmonary lesions indicated that the tissue reaction was primarily related to the particles. The pulmonary damage following aspiration of a nonparticulate antacid has been shown to be less severe than that following aspiration of a particulate type.[5]

The introduction of H_2-receptor antagonists has made available an effective method of reducing the volume and increasing the pH of the gastric contents. In obstetric patients the most usual means of achieving these objectives entails oral administration of the drugs to all patients throughout labour. However, only a small proportion of patients in labour are at risk from aspiration pneumonitis, as the majority do not require general anaesthesia for delivery. Ideally a simple regimen of antacid prophylaxis is required which will effectively treat all patients at risk of aspiration pneumonitis, but only those patients.

For the past year, administration of oral antacids at regular intervals to patients in labour at the Queen Mother's Hospital, Glasgow, has ceased, and the following regimen instituted. When the decision is made to effect delivery by emergency Caesarean section, cimetidine 200 mg is administered intramuscularly. In the operating theatre, and shortly before induction of anaesthesia, 30 ml of 0.3 M sodium citrate is given

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orally. This policy is followed in all patients, irrespective of the type of anaesthesia employed. The objective of this study was to determine what proportion of patients to whom this regimen had been administered, had an intragastric pH of less than 2.5 at the time of induction of general anaesthesia.

Methods

One hundred patients who required delivery by emergency Caesarean section under general anaesthesia were treated as described. General anaesthesia was induced in those patients who expressed a preference for this type of anaesthetic, or in whom it was considered that insufficient time was available to establish a regional block. Seventeen patients had received narcotic analgesia and a further 22 patients an epidural block for pain relief in labour.

The technique of general anaesthesia has been described in detail,[6] and consisted of lateral tilt, pre-oxygenation, cricoid pressure, thiopentone and suxamethonium, followed by tracheal intubation. An 18-gauge Salem sump tube was inserted following induction of anaesthesia and the gastric contents aspirated. An attempt was made to empty the stomach by repeated aspiration as the tube was withdrawn. The total volume aspirated was noted and, shortly before the end of surgery, repeat aspiration was made and again the volume noted. The samples were transferred to the laboratory for measurement of the pH by meter.

The duration of labour and the interval between the administration of cimetidine and the induction of anaesthesia were noted. Analysis of variance was done for the pH of the first gastric samples obtained and for the natural logarithm transformation of the gastric volumes, performed to normalise the data as required for analysis of variance. Pearson's *r* correlation was used as required where bivariate normality was seen to be acceptable.

Table 1. Range and median value (minutes) of time interval between injection of cimetidine and induction of general anaesthesia, and mean pH values of first and second gastric samples obtained.

Time interval range (minutes)	5–208
Median time interval (minutes)	52.5
Mean pH of first gastric samples (SD)	6.58 (0.95)
Mean pH of second gastric samples (SD)	6.7 (0.99)

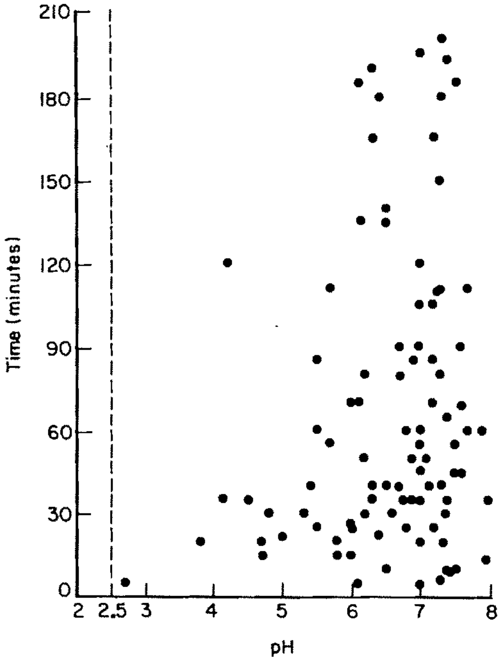

Fig. 1. Relationship between the time interval from injection of cimetidine to induction of anaesthesia, and pH of the gastric sample obtained.

Results

The mean pH of the first samples was 6.58 (Table 1), and only one sample had a recorded pH of less than 3 (Fig. 1). The lowest pH recorded was 2.7. The mean pH of the second samples was similar to that of the first.

Figure 1 shows the absence of any clear relationship between the time interval from injection of cimetidine to obtaining the gastric sample following induction of anaesthesia, and its pH. The range of time intervals was 5–208 minutes, with a median of 52.5 minutes. Anaesthesia was induced in 27 patients within 30 minutes of receiving cimetidine, and the two lowest pH values were observed in this group.

Twenty-two patients delivered by Caesarean section had not been in labour. The type of analgesia administered during labour, the proportion of patients receiving it and the gastric volumes aspirated from all patients are shown in Table 2. The differences in gastric volumes were significant in patients who were not in labour or who had received epidural analgesia alone, when compared with patients who had received both sedation and epidural analgesia ($p < 0.05$). Thirty-five patients had a gastric

Table 2. Mean and standard deviation of pH of the first gastric samples and of the natural logarithms of the gastric volumes, characterised by the method of sedation. Figures in parentheses are the mean gastric volumes. The differences among the gastric volumes were significant (*F* value 2.522, $p < 0.05$), but not among their pH values (analysis of variance).

	No sedation ($n = 39$)	Not in labour ($n = 22$)	Sedation only ($n = 17$)	Sedation and epidural ($n = 6$)	Epidural ($n = 16$)
Gastric volumes					
Mean natural logarithm (volume in ml)	3.24 (26)	2.58 (13)	3.11 (22)	3.48 (32)	2.59 (13)
SD	0.93	1.1	1.01	1.35	1.2
pH of first gastric sample					
Mean	6.26	6.81	6.81	6.78	6.98
SD	1.11	0.92	0.68	0.78	0.53

volume of less than 30 ml, and no significant correlation was noted between gastric volume and duration of labour in the sedated patients ($r = 0.144$, $p > 0.05$).

Discussion

The injection of cimetidine intramuscularly and the administration of oral sodium citrate preceding induction of anaesthesia, effectively maintained the pH of the gastric contents above 2.5. Taylor and Pryse-Davies[2] noted that 43% of patients who had not received antacid therapy had a gastric pH of less than 2.5: the use of oral antacids reduces this to 5–20% depending on the choice of agent, volume, concentration and timing of its administration. Failure to reduce maternal mortality from acid aspiration despite the almost universal adoption in the UK of oral antacid therapy in labour, has usually been ascribed to failure of administration or failure to mix with the gastric contents. However, since Gibbs *et al.*[4] demonstrated pneumonitis following aspiration of a particulate antacid (a mixture of aluminium and magnesium hydroxides), failure to prevent obstetric mortality in the UK has been speculatively ascribed to the particulate nature of magnesium trisilicate; speculatively because magnesium trisilicate is slightly different from the antacid studied by Gibbs.[7]

It is widely accepted that aspiration of acid gastric contents with a pH of less than 2.5 will produce a pneumonitis, but the volume required to produce a fatal pneumonitis is obviously critical. The volume in humans remains speculative, although animal work suggests that 0.4 ml/kg is the maximum volume of aspirate which will not produce significant lung changes on inhalation.[5,8] Clearly, the routine administration of oral antacids during labour, particularly to sedated patients with reduced gastric emptying rates, must increase the volume of the gastric contents and the attendant risks.[9] H_2-receptor antagonists effectively reduce gastric volumes and acidity, but they require a period of time to achieve first a pharmacologically active blood concentration, and then to exert an inhibiting effect. They cannot affect the pH of the stomach contents present at the time of their administration and, consequently, their use has been confined to patients in labour and those undergoing elective Caesarean section.[10–12]

The introduction of ranitidine has overcome the disadvantage of the shorter duration of action of cimetidine and the need for frequent administration, but it is slower in onset. As presently practised, antacid therapy using these drugs requires regular administration during labour. Timing may be critical, and their administration may be subject to delays or errors. It can be calculated that, for England and Wales, if antacid therapy is routinely prescribed to all patients in labour, 800 000 patients will receive it in the hope of preventing the death of four, and approximately 700 000 patients will have received treatment for a disorder from which they have not been at risk as general anaesthesia for delivery was not required. Such a drug requires an enviable and formidable record of safety, and it is not yet clear whether H_2-receptor antagonists provide this, although present reports are favourable.[13]

Effective antacid prophylaxis was also required for the 22 patients who were not in labour when the decision to undertake delivery by Caesarean section was made. Furthermore, 27 patients were delivered within 30 minutes of the decision being made, and not all of these patients were in labour. In these circumstances a combination of H_2-receptor antagonist and oral therapy is required. It might be argued that the use of cimetidine was unnecessary, but from Fig. 1 it can be seen that the cimetidine would be fully effective in approximately 50% of patients and have a substantial but diminishing effect in the remainder. Intravenous administration would result in a faster onset of action, but adverse cardiovascular responses have been reported if cimetidine is injected rapidly by this route.[14]

In conclusion, the use of a combination of cimetidine and sodium citrate for emergency obstetric general anaesthesia proved to be safe and effective. It has the added advantage of treating only those patients at risk of acid aspiration; it removes from the risks of exposure to drugs and their consequent errors of administration, the vast majority of obstetric patients and their babies who do not require surgical intervention. It has been enthusiastically accepted by the nursing staff, partly as a result of the reduction in the number of patients required to swallow unpalatable medication, but also because of the reduction in the logistical burden of accurate administration of routine antacid therapy to all patients in labour in a busy obstetric unit.

Acknowledgments

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Recovery from propofol infusion as the main agent for outpatient arthroscopy

A comparison with isoflurane

W. W. A. ZUURMOND, L. VAN LEEUWEN AND J. H. J. H. HELMERS

Summary

Propofol by continuous intravenous infusion has been compared with isoflurane as the main anaesthetic agent for outpatient arthroscopy of the knee. In 40 unpremedicated patients, anaesthesia was induced with propofol 2 mg/kg and vecuronium bromide 0.1 mg/kg and maintained after tracheal intubation with nitrous oxide 66% in oxygen. One group received 3% isoflurane prior to intubation and 0.9% during maintenance, while the other received a continuous intravenous infusion of propofol at a rate of 10 mg/kg/hour. Recovery was assessed by the time to opening eyes, to be able to answer five questions correctly, to recovery of ocular balance (Maddox Wing test) and by comparing pre- and postoperative performance in a paper and pencil test (the p-deletion test). After 3 hours all the patients were fit for discharge. Recovery tests showed no differences between the groups. All patients were satisfied with the anaesthesia. Full recovery took on average 1.5 days (range between 1 hour and 14 days) in both groups. Patients' opinion 1 month after the procedure should be included in every study concerning recovery. Anaesthesia by continuous propofol infusion results in quick recovery comparable with that following isoflurane anaesthesia.

Key words

Anaesthetics, intravenous; propofol.
Anaesthetics, inhalational; isoflurane.

Propofol (2,6-di-isopropylphenol) formulated as an oil emulsion is a new, short-acting hypnotic with a rapid onset of action.[1] Pharmacokinetic and clinical studies suggest that this drug, by virtue of its high clearance and short elimination half-life, might be especially suitable for day case anaesthesia.[2] This study was designed to compare recovery from propofol–isoflurane and propofol–continuous propofol intravenous infusion anaesthesia in day case patients undergoing arthroscopy of the knee under general anaesthesia.

Methods

Forty patients aged 16–48 years, of ASA class 1, scheduled for diagnostic arthroscopy of the knee in the day case surgery unit, were randomly allocated to two groups (A and B) of 20. to receive general anaesthesia. None was taking

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any medication to affect recovery from anaesthesia and all gave informed consent.

After assessment, baseline pre-anaesthetic values of tests used to assess recovery were obtained. No premedication was given. After pre-oxygenation, anaesthesia was induced in all patients with propofol 2 mg/kg followed by vecuronium bromide 0.1 mg/kg. Anaesthesia was maintained with nitrous oxide 66% in oxygen, using a circle and absorber with a total fresh gas flow of 3 litres/minute, and the patients' lungs were ventilated to maintain $F\text{E}'\text{CO}_2$ between 4 and 5%.

Patients in group A received isoflurane 3% prior to tracheal intubation and isoflurane 0.9% (inspired) during maintenance of anaesthesia. Patients in group B received, after induction of anaesthesia, a continuous intravenous infusion of propofol at a rate of 10 mg/kg/hour.

The isoflurane and nitrous oxide in group A and the propofol infusion and nitrous oxide in group B were stopped at the end of surgery. This moment was defined as the end of anaesthesia. Neuromuscular blockade was reversed with atropine and neostigmine.

No other drugs were given before or during anaesthesia.

Assessment of recovery

Recovery from anaesthesia was assessed using the same criteria as in a previous study:[3] the time elapsed from the end of anaesthesia until spontaneous eye opening; the time elapsed from eye opening until the patients were orientated and could answer the following questions: What is your name? How old are you? Can you show me your right thumb? What day is it today? What is 7 × 8? Measurement of ocular imbalance was assessed by a Maddox Wing test[4] every 20 minutes. The time between the end of anaesthesia and the moment that the patients reached their pre-anaesthetic values was measured. A p-deletion test was performed 30, 60 and 120 minutes after the end of anaesthesia, using a modified form of the test described by Dixon and Thornton.[5] Every patient received a text containing 140 p's and was asked to delete as many p's as possible without omissions in 3 minutes. The total number of p's deleted was measured.

Clinical assessment was made during the recovery period with regard to level of arousal, the incidence of side effects and the need for antiemetic or analgesic drugs. The patients' opinion of the anaesthetic was sought 1 month later.

Statistical analysis was performed using paired and unpaired Student's t-tests; $p < 0.05$ was considered to be significant.

Table 1. Patient age, weight and sex ratio in the two groups, with duration of anaesthesia: mean (SEM).

	Group A ($n = 20$; 10M, 10F)	Group B ($n = 20$; 11M, 9F)
Age, years	28.3 (1.3)	31.9 (2.2)
Weight, kg	74.7 (2.3)	71.4 (2.8)
Duration, minutes	49.9 (1.8)	51.4 (2.9)

Results

Table 1 shows the mean age, weight, sex ratio and duration of anaesthesia in the two groups. All patients underwent anaesthesia and surgery without complications. The time between the end of anaesthesia and eye opening, and the time to achieve correct verbal responses, did not differ between groups A and B (Table 2). There was also no difference between the groups in the time to recovery of ocular balance using the Maddox Wing test. The range was 30–150 minutes (Table 2).

Table 2. Mean (SEM) values of three indices of recovery.

Recovery index	Group A ($n = 20$) (propofol–isoflurane)	Group B ($n = 20$) (propofol–propofol)	
Time to opening eyes, minutes	8.4 (0.6)	8.9 (0.8)	NS
Time to giving correct answers to 5 questions *after* eye opening, minutes	2.4 (0.3)	2.8 (0.4)	NS
Time to recovery of ocular imbalance (Maddox Wing) from end of anaesthesia, minutes	84.5 (8.5)	90.8 (6.7)	NS

NS, not significant.

Table 3. Results of p-deletion test, with statistical analysis. The groups were compared with their own pre-anaesthetic control values.

	Number of deleted p's in text	
	Group A ($n = 20$) (propofol–isoflurane)	Group B ($n = 20$) (propofol–propofol)
Pre-operative	62.5 (3.4)	56.9 (2.5)
30 minutes after end of anaesthesia	44.9 (3.7) **	40.5 (3.2) *
60 minutes after end of anaesthesia	62.7 (3.3)	55.2 (2.9)
120 minutes after end of anaesthesia	68.0 (3.3)	64.3 (3.6)

* $p < 0.01$. ** $p < 0.001$.

Table 4. The incidence of side effects and postoperative drugs administered during the recovery period.

	Number of patients	
	Group A (propofol–isoflurane)	Group B (propofol–propofol)
Postoperative drugs		
Neostigmine (1 mg), atropine (0.5 mg) intravenously	8	8
Antiemetic	0	0
Analgesic	3	4
Side effects		
Nausea and/or vomiting	0	0
Dizziness, headache	1	0
Not fit for discharge	0	0

The p-deletion scores reached their pre-anaesthetic values in patients in both groups in 60 minutes (Table 3). The two groups showed a similar pattern of recovery in the p-deletion scores; there were no significant differences between the values pre-operatively or at 30, 60 and 120 minutes. Clinical assessment of wakefullness in both groups showed that after 60 minutes all patients were completely awake.

The incidence of side effects and the drugs given postoperatively are listed in Table 4. None of the patients suffered from nausea and/or vomiting. All patients were fit for discharge at the end of a recovery period of 3 hours.

At the 1-month interview, all patients stated that they were satisfied with the anaesthetic they had received. The mean time for full recovery ('feeling back to normal') was 1 day (range 1 hour–14 days) for both groups.

Two patients in group A spontaneously mentioned hair loss after the procedure. The surgeon was satisfied with the operative conditions in both groups.

Discussion

Assessment of recovery should include a number of tests: the time of immediate awakening and orientation of the patient; measurement of ocular imbalance by the Maddox Wing test as described by Korttila[6] (this test is easy to perform; discharging a patient with ocular imbalance could be dangerous); a test of short duration (such as a paper and pencil test) which will not influence the normal recovery period (too intensive testing could awaken the patient artificially); and an interview or questionnaire 1 month after anaesthesia. The patient can offer a lot of information about his experiences after discharge. Every study that concerns recovery should include the patient's opinion about his functioning at home and/or at work.

A standardised anaesthetic technique was employed to compare recovery from anaesthesia using propofol infusion or isoflurane as the main short-acting agent. Propofol was given for induction in both groups, also vecuronium bromide, nitrous oxide and oxygen.

Propofol as a continuous intravenous infusion was administered at a rate of 10 mg/kg/hour, which resulted in smooth anaesthesia. Major *et al.*,[7] who used the Cremophor formulation of propofol, recommended an initial infusion rate of 12 mg/kg/hour, while others used infusion rates of 7–8 mg/kg/hour.[8] Many anaesthetists have stressed the ease of administration if an intravenous technique is used; our dose scheme merely simplifies calculations.

Isoflurane was administered to the patients, but there is little difference in recovery times in outpatients as compared to halothane and enflurane.[9] In our study there were no differences between propofol infusion and isoflurane with regard to the recovery period. Both anaesthetic techniques provided satisfactory recovery within 3 hours in all cases. Awareness did not occur. The hair loss in two patients in the isoflurane group could be coincidental; however, in a previous study one patient complained of hair loss after methohexitone–isoflurane anaesthesia.[3] Attention should be paid to this possible side effect in any follow-up study after anaesthesia.

We conclude that anaesthesia with propofol infusion as the main agent results in a quick recovery comparable with that following isoflurane. Continuous intravenous anaesthesia offers advantages over inhalational anaesthesia by avoiding harm to theatre personnel that might result from theatre pollution.

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Anaesthesia, 1987, Volume 42, pages 360–365

Propofol combined with nitrous oxide–oxygen for induction and maintenance of anaesthesia

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Summary

After a bolus of 2 mg/kg, propofol was given by continuous infusion (150 µg/kg/minute for 30 minutes and then 100 µg/kg/minute) supplemented with nitrous oxide for anaesthesia during ear surgery in 12 patients. Cardiovascular changes were not significant except for a decrease in heart rate after 60 minutes. Acid-base balance was unaffected by the amount of fatty emulsion. Cortisol levels showed a nonsignificant decrease during the prolonged administration of propofol but had recovered completely by one hour following anaesthesia. Mean blood concentrations of propofol were 10.5 µg/ml (SEM 1.2) at the onset of unconsciousness, between 3.4 and 4.5 µg/ml during continuous infusion and 2.9 µg/ml (SEM 0.3) on awakening. Patients opened their eyes 6 minutes (SEM 1) after discontinuation of the infusion, and were responsive at 7.5 minutes (SEM 0.5), which suggests that propofol infusion can be used safely for surgery of 2 hours' duration.

Key words

Anaesthetics, intravenous; propofol.

Propofol (2,6-di-isopropylphenol) was first used in the Cremophor formulation by Kay and Rolly[1] and shown to possess many suitable characteristics of an intravenous induction agent. Recently, propofol became available in an aqueous emulsion formulation (Diprivan). The clinical value of propofol has been reassessed with this formulation. Studies of its pharmacokinetic properties, induction characteristics and interactions with other drugs, suggest a role for propofol in intravenous anaesthesia, with or without nitrous oxide supplementation.

This study reports our experience with propofol in emulsion formulation for the induction and maintenance of anaesthesia supplemented with nitrous oxide and oxygen. The objective was to assess the effect of continuous intravenous administration of propofol on the quality of anaesthesia, cardiovascular responses, arterial blood gases and acid-base balance, metabolic variables and liver enzymes. Blood concentrations of propofol were measured on a serial basis and compared with electromyographic and electroencephalographic recordings.

Methods

The study involved 12 informed patients (seven females, five males). All were ASA grade 1 or 2, between 18 and 50 years old and were

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undergoing ear surgery. The study was approved by the ethical committee of the hospital. The patients were premedicated with fentanyl 0.1 mg, droperidol 5 mg and atropine 0.5 mg intramuscularly given 30 minutes before anaesthesia. Upon arrival in the operating theatre, patients' lungs were oxygenated whilst the monitoring devices were applied.

Throughout each procedure, ECG lead II was monitored continuously and arterial blood pressure recorded intermittently with an automatic blood pressure monitor (Dinamap). Two pick-up electrodes and one standard electrode were applied frontotemporally for the registration of frontal electromyogram (EMG) and electroencephalogram (EEG) by means of the anaesthesia and brain activity monitor (ABM Datex). Neuromuscular transmission was assessed by the neuromuscular transmission monitor incorporated in the ABM.

A fast-running infusion of saline 0.9% was given via an antecubital vein. Throughout the procedure 500 ml of this solution was infused. A second intravenous catheter was sited in the contralateral arm for intermittent blood sampling. Anaesthesia was induced with propofol 2 mg/kg given over 20 seconds into the fast-running infusion. When the patient was asleep, suxamethonium 1 mg/kg was given to allow tracheal intubation. The patients' lungs were ventilated mechanically with a mixture of nitrous oxide and oxygen (2:1), and $P\text{aco}_2$ was maintained between 4.3 and 5.0 kPa. The operation field was infiltrated with 3–4 ml lignocaine 2% containing adrenaline 1:80 000 before surgical incision.

Anaesthesia was maintained with an infusion of propofol started immediately after induction. The initial rate of 150 μg/kg/minute was reduced to 100 μg/kg/minute after 30 minutes. Increments of vecuronium or pancuronium were given for muscle relaxation. At the end of the procedure the infusion was stopped and 100% oxygen was given.

Blood samples for determination of blood sugar and cortisol were taken pre-operatively, one hour after the infusion had been started, and one hour after the end of the procedure. Acid-base balance and blood gases were measured 5 and 60 minutes after the start of induction. Liver enzymes, SGOT, SGPT and alkaline phosphatase, were determined pre-operatively and 1 and 3 days postoperatively. The concentration of propofol in venous blood was measured at the time of falling asleep, every 15 minutes during the procedure, at the time of awakening, when patients were responsive and one hour postoperatively. Concentrations were determined by high-performance liquid chromatography with fluorescence detection.

Statistical analysis was performed by the Freedman two-way analysis of variance between the groups. When values were missing, the Kruskal–Wallis one-way analysis of variance by ranks was applied.

Results

The mean age of the patients was 28 years (SEM 3) and the mean weight 67 kg (SEM 2). Induction of anaesthesia was successful and smooth in all patients. Depth of anaesthesia was clinically acceptable throughout procedures lasting for a mean of 120 minutes (SEM 15, range 70–180 minutes). The cardiovascular changes are shown in Fig. 1. There was a decrease in the systolic and diastolic blood pressures 15 minutes after induction of anaesthesia, but the changes were not statistically significant. Heart rate decreased during the first 45 minutes of infusion and was statistically significantly lower ($p < 0.02$) from 60 minutes onwards, but remained stable during the rest of the procedure (Fig. 1).

The values for acid-base balance and blood gases, assessed 5 and 60 minutes after the start of anaesthesia, were within normal ranges and did not show any statistically significant change (Table 1).

Cortisol and blood sugar levels, measured before induction, one hour after the start of anaesthesia and one hour after the end of the procedure, were within normal ranges and with no statistically significant difference between values (Table 2, Fig. 2). Liver enzymes were determined pre- and postoperatively. There was no change in SGPT. Three days postoperatively SGOT increased ($p < 0.05$) and alkaline phosphatase decreased ($p < 0.05$) (Table 2, Fig. 2).

The mean blood concentration of propofol was 10.5 μg/ml (SEM 1.2) at the onset of unconsciousness. This was significantly higher ($p < 0.001$) than all values recorded during and after infusion. The mean blood concentrations during continuous infusion were within the range 3.4–4.5 μg/ml. They showed a statistically significant decrease 15 minutes after the

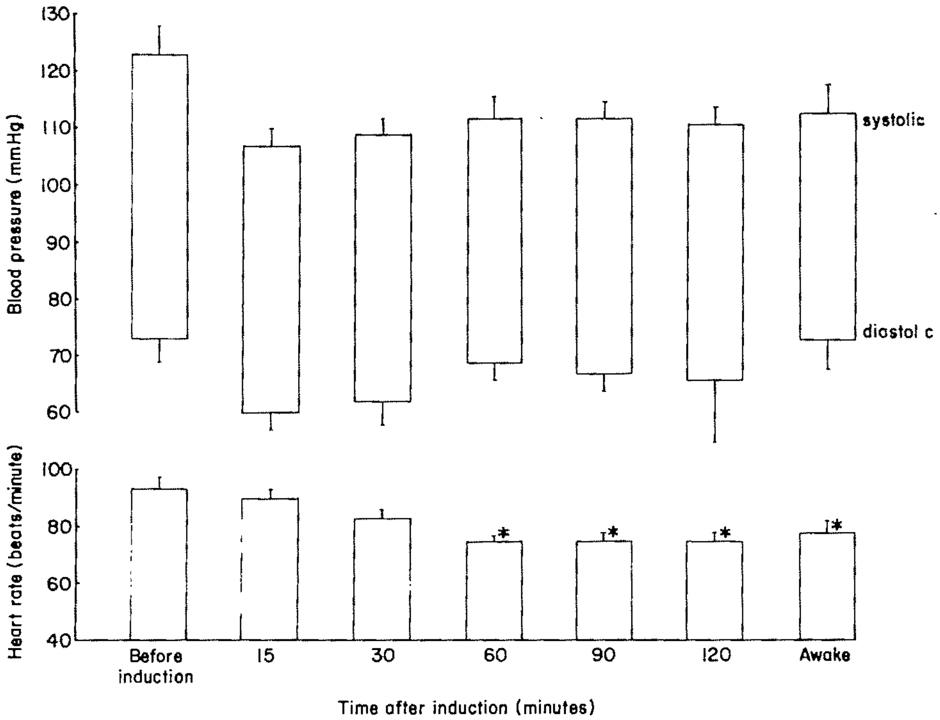

Fig. 1. Changes in blood pressure and heart rate in 12 patients. Mean and SEM values are shown. *Significant difference ($p < 0.02$) from values before induction and 15 minutes after induction.

Table 1. Acid-base balance and blood gases. Values expressed as mean (SEM); $n = 12$.

	pH	$Pa\text{CO}_2$, kPa	Standard bicarbonate, mmol/litre	Base excess, mmol/litre	$Pa\text{O}_2$, kPa
5 minutes after start of anaesthesia	7.37 (0.01)	5.07 (2.27)	23 (0.3)	−2.3 (0.4)	22.3 (1.73)
60 minutes after start of anaesthesia	7.38 (0.01)	4.67 (1.2)	22.9 (0.3)	−2.6 (0.4)	17.5 (1.73)

Table 2. Cortisol levels, blood sugar, liver enzymes and alkaline phosphatase. Values expressed as mean (SEM); $n = 12$.

	Cortisol, µmol/litre	Blood sugar, mmol/litre
Pre-operatively	0.66 (0.08)	5.11 (0.28)
One hour after start of continuous infusion	0.47 (0.08)	6.22 (0.39)
One hour postoperatively	0.61 (0.11)	5.66 (0.28)

	SGOT, U/litre	SGPT, U/litre	Alkaline phosphatase, U/litre
Pre-operatively	12 (1) }	11 (1)	176 (40) }
One day postoperatively	12 (1) } * } *	9 (1)	165 (36) } * } *
Three days postoperatively	20 (3) }	12 (1)	139 (31) }

* $p < 0.05$.

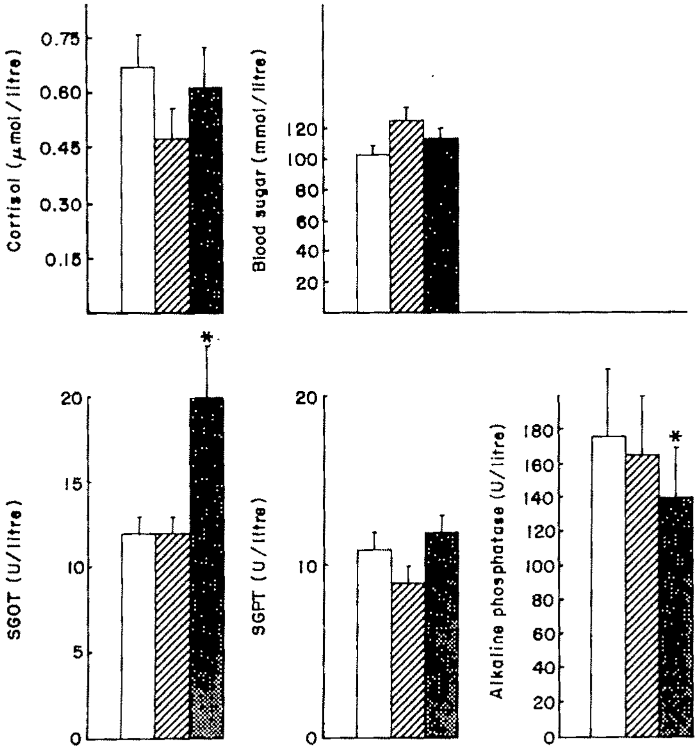

Fig. 2. Cortisol, blood sugar, liver enzymes and alkaline phosphatase in 12 patients. Mean and SEM values are shown. *Significant difference ($p < 0.05$). *Cortisol and blood sugar*: □, pre-operative levels; ■, one hour after start; ▨, one hour postoperatively. *Liver enzymes and alkaline phosphatase*: □, pre-operative levels; ■, 1st day postoperatively; ▨, 3rd day postoperatively.

infusion rate was decreased from 150 to 100 μg/kg/minute, but increased with further administration to values similar to those recorded at the beginning of infusion.

From the end of infusion and discontinuation of nitrous oxide, it took 6 minutes (SEM 1) for patients to open their eyes and 7.5 minutes (SEM 0.5) before they were responsive. The corresponding blood concentrations were 2.9 μg/ml (SEM 0.3) and 1.8 μg/ml (SEM 0.2), respectively. Each value was significantly lower than that in the preceding sample. The blood concentration showed a further significant decrease to 0.8 μg/ml (Fig. 3) one hour after anaesthesia.

During induction, EMG values decreased abruptly to 10–20% of their initial values and remained stable at this low level throughout the infusion; they returned to the original values at the time of awakening. EEG waves showed a slower frequency pattern and some increase in amplitude, particularly at the early stage of the propofol infusion. The EEG frequency increased markedly when the infusion was stopped.

Discussion

A clinically smooth induction of anaesthesia with propofol was confirmed by ABM assessments. Changes in objective parameters, such as EMG and EEG, were noted shortly before patients were clinically asleep. This has also been reported by Kay.[2] In our study, EMG and EEG frequency decreased before the eyelash reflex was abolished.

The haemodynamic effects of an induction dose of propofol have already been studied.[3–5] In agreement with reported observations, we noted a decrease in systolic and diastolic blood pressures but these changes were not, however, statistically significant. This may be ascribed to the effect of intubation and the start of controlled ventilation. In the studies of other investigators, patients' tracheas were not intu-

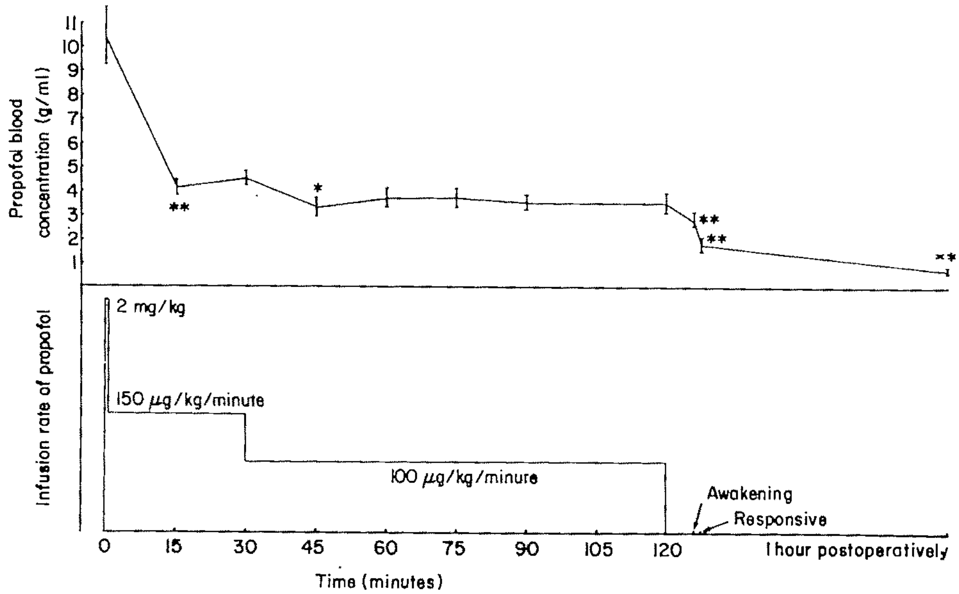

Fig. 3. Propofol blood concentrations related to rate of infusion in 12 patients. Mean and SEM values are shown. * Significant difference ($p < 0.05$) from previous value; **$p < 0.02$.

bated and they breathed spontaneously. There was a reduction in heart rate when the infusion rate was decreased after 30 minutes.

The choice of muscle relaxant appeared to have no effect on cardiovascular variables, as no differences were apparent between those who received pancuronium (ten patients) and vecuronium (two patients).

Taking into account the total amount of propofol in fatty emulsion which was administered (98 ml, SEM 17), it is important that there was no change in acid-base balance or blood gases. Standard bicarbonate and base excess were unchanged one hour after the start of infusion in the group as a whole, and in the three patients monitored for 3 hours.

The effect of propofol on cortisol levels was examined, as a major drawback to the use of etomidate by continuous infusion is the significant decrease in plasma cortisol that it produces. A marked, but nonsignificant, decrease in cortisol levels was observed after one hour of administration of propofol. This observation is in contrast to the results of Doenicke[6] and Kenyon *et al.*,[7] but Kay *et al.*[8] have suggested that propofol might influence plasma cortisol after administration for 30 minutes. One hour after the end of the procedure, however, cortisol levels had reapproached pre-induction values, as was reported by Kay *et al.*[8] 3 hours after induction.

Blood sugar values were unchanged throughout the procedure. There was no change in liver enzymes on the first day after anaesthesia, but unexpectedly large changes (still within physiological range, however) were observed 3 days postoperatively. These results are in contrast to those of Robinson and Patterson.[9] Since the differences cannot be related to the administration of propofol, we suggest the influence of other factors such as nutrition or bone fraction resorption.

After a loading dose of 2 mg/kg, the mean blood concentration of propofol at the onset of unconsciousness was 10.5 μg/ml (SEM 1.2). During the first 30 minutes of the infusion, a steady state level was achieved and was maintained after reduction of the initial rate of infusion. These findings are compatible with smooth clinical anaesthesia. Individual blood concentrations were not correlated with age, sex or weight.

As patients opened their eyes only 6 minutes (SEM 1) after nitrous oxide and propofol were stopped, we suggest that the infusion is continued until the end of the procedure to prevent awareness.

It is concluded that induction of anaesthesia with propofol together with maintenance by an infusion to supplement nitrous oxide in paralysed and ventilated patients, provides adequate levels of anaesthesia for up to 2 hours in premedicated patients.

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Comparison of etomidate and propofol for anaesthesia in microlaryngeal surgery

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Summary

Propofol and etomidate were compared as hypnotics in total intravenous anaesthesia for microlaryngeal surgery combined with jet ventilation. Two groups of 15 patients were studied. In group 1, propofol 2.0 mg/kg was used for induction. For maintenance a continuous infusion of 12 mg/kg/hour was used for the first 10 minutes, followed by 9 mg/kg/hour for the next 10 minutes and 6 mg/kg/hour thereafter. In group 2, the induction dose of etomidate was 0.3 mg/kg followed by continuous infusion of 1.8 mg/kg/hour for 10 minutes, 1.5 mg/kg/hour for the next 10 minutes and 1.0 mg/kg/hour thereafter. Alfentanil was given for analgesia and suxamethonium for muscle relaxation. The propofol group showed better surgical conditions, more stable anaesthesia and better recovery according to the Steward score. Recovery times to opening eyes on command were comparable for both groups.

Key words

Anaesthetics, intravenous; propofol, etomidate.
Surgery; microlaryngeal.

Anaesthesia for microlaryngeal procedures requires a special approach. On the one hand, the surgeon wants an unhindered view of the vocal cords and the upper part of the trachea, while on the other, the anaesthetist has to provide adequate ventilation. Moreover, microlaryngeal surgery necessitates a deep level of anaesthesia to prevent vocal cord movement and other reactions to surgical stimuli. Recovery from anaesthesia must be rapid to prevent possible aspiration of blood or debris.[1]

Jet ventilation is accepted as the best ventilation technique for microlaryngeal surgery. Total intravenous anaesthesia in combination with jet ventilation is recommended as the technique of choice for vocal cord surgery and other procedures in the upper airways.[2–5] The combination of inhalational anaesthesia and jet ventilation is difficult. The lack of equipment for accurate administration of inhalational anaesthetics as well as the heavy pollution with waste anaesthetic gases and vapours (directly into the face of the surgeon) are problems still to be solved.

Several drugs can be used for total intravenous anaesthesia. The combination of etomidate, fentanyl and suxamethonium has been recommended for microlaryngeal surgery[3–5] and has been used in our hospital for several years. After the introduction of alfentanil, fen-

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tanyl was replaced in our technique by this new short-acting narcotic. Propofol (2,6-di-isopropylphenol in an emulsion of soyabean oil) recently became available as an hypnotic agent. It has been demonstrated to have good properties for induction as well as for maintenance of anaesthesia.[6-12]

The aim of this study was to investigate whether propofol has advantages over etomidate for microlaryngeal procedures.

Methods

Thirty patients of ASA class 1–3, aged between 20 and 79 years, scheduled for microlaryngeal procedures, were studied. They had no history of allergy to the drugs to be used or evidence of atopy. No patient with a history of serious hepatic, renal, haematological or metabolic disease was included. All patients gave their informed consent before participating in the study, which was approved by the hospital ethics committee. The patients were premedicated with atropine 0.5 mg intramuscularly approximately 30 minutes before induction. Prednisolone 25 mg was added to the premedication to prevent postoperative laryngeal oedema.

On arrival in the operating theatre, an indwelling cannula was inserted in a large vein on the dorsum of the hand or forearm and connected to an infusion of Ringer's solution. The electrocardiogram and finger plethysmogram were continuously monitored. Arterial pressure was measured using an automatic non invasive blood pressure monitor (Dinamap). The baseline values of arterial pressure and heart rate were measured before induction and subsequent measurements were made 2 minutes after induction and at 5-minute intervals thereafter.

The patients were randomly allocated to two groups of 15 patients each. In group 1, propofol 2 mg/kg was used for induction. For maintenance a continuous infusion of the same agent was given: 12 mg/kg/hour for the initial 10 minutes, followed by 9 mg/kg/hour for another 10 minutes and thereafter 6 mg/kg/hour. In group 2, anaesthesia was induced with etomidate 0.3 mg/kg followed by continuous infusion of 1.8 mg/kg/hour for the first 10 minutes, 1.5 mg/kg/hour for the next 10 minutes and 1.0 mg/kg/hour thereafter. Both drugs were administered by a calibrated syringe pump. Propofol was given in undiluted form (10 mg/ml) and etomidate was used in a concentration of 2 mg/ml (4 ml of 125 mg etomidate base per ml of ethyl alcohol diluted to 250 ml with normal saline).

In both groups a bolus dose of alfentanil 0.5–1 mg was administered 60 seconds prior to induction. Further bolus doses of alfentanil 0.5–1 mg were given on signs of painful reactions to surgical stimuli. Topical anaesthesia of the larynx and vocal cords was performed with lignocaine 4% spray with a maximum volume of 3 ml. Suxamethonium was given to facilitate tracheal intubation and incremental bolus doses of 25 mg were given when needed. Jet ventilation was performed with 100% oxygen via a CH14 nasotracheal catheter using an Accutronic high frequency jet ventilator.

Onset of anaesthesia was defined in two ways: firstly, as the interval between start of administration of the hypnotic agent and cessation of counting; and secondly, as the interval between start of administration of the hypnotic agent and disappearance of the eyelash reflex. Two recovery times were also defined: the interval from cessation of infusion until opening eyes on command, and that until correct response to simple questions. Recovery from anaesthesia was assessed by the Steward score[13] at 5, 15 and 30 minutes after the first response to opening eyes on command.

All side effects were recorded from induction of anaesthesia up to 24 hours postoperatively. The intensity of pain on injection was recorded as being mild, moderate or severe, depending on the response of the patient to questioning: mild when minor sensations were noted, moderate if discomfort was admitted on request, and severe if discomfort was indicated spontaneously. Depth and smoothness of anaesthesia were evaluated postoperatively with regard to the lack of, or need for, additional bolus doses of alfentanil and suxamethonium.

The statistical calculations were carried out using the SPSS routine package (SPSS Inc., Chicago, Illinois). Analysis of variance was used as the statistical test to analyse differences between groups. Comparisons of incidences were performed by the Chi squared test or Fisher's exact test (in a 2 × 2 table). Student's *t*-test was used for paired observations. In statistical tests $p < 0.05$ was considered significant.

Table 1. Demographic data. Values expressed as mean (SD).

	Propofol (*n* = 15)	Etomidate (*n* = 15)	
Age, years	55.5 (13.2)	54.9 (12.7)	NS*
Weight, kg	77.1 (9.9)	72.6 (14.8)	NS
Height, cm	171.5 (10.5)	173.0 (8.8)	NS
Duration of surgery, minutes	12.5 (15.3)	16.6 (10.4)	NS
Duration of anaesthesia, minutes	16.6 (15.0)	21.3 (10.6)	NS

* Analysis of variance. NS, Not significant.

Table 2. Induction and recovery times. Values expressed as mean (SD).

	Propofol (*n* = 15)	Etomidate (*n* = 15)	
Stop counting, seconds	39.1 (8)	36.9 (11.5)	NS*
Negative eyelash reflex, seconds	52.4 (11)	49.1 (12.8)	NS
Eyes open on command, minutes	6.9 (2.5)	6.4 (2.6)	NS
Reply on questioning, minutes	7.3 (4.2)	12.4 (9.4)	NS

* Analysis of variance. NS, Not significant.

Table 3. Total doses of suxamethonium and alfentanil during anaesthesia. Values expressed as mean (SD).

	Propofol (*n* = 15)	Etomidate (*n* = 15)	
Suxamethonium, mg/kg	1.8 (0.9)	2.6 (0.6)	$p < 0.005$*
Alfentanil, μg/kg	16 (8)	20 (8)	$p < 0.13$

* Analysis of variance.

Results

Both groups were comparable for age, height, weight and duration of surgery and anaesthesia (Table 1). Induction was accomplished in all patients with the induction bolus dose. Apnoea was observed in all patients after administration of the induction dose. Duration of apnoea was not measured because a muscle relaxant was administered immediately after induction.

Mild pain on injection occurred in one patient in the propofol group and in two patients given etomidate. Five patients in the etomidate group showed myoclonus during induction and one patient in this group had hiccoughs. In the propofol group induction was smooth, with no side effects. Approximately 40 seconds after the start of induction patients stopped counting, followed 10 seconds later by disappearance of the eyelash reflex. There was no significant difference between the two groups in either induction time (Table 2).

Muscle relaxants were given during the maintenance period, as indicated by return of vocal cord movement. Six patients in the propofol group and only one patient in the etomidate group needed no further muscle relaxation during the procedure. The total dose of suxamethonium in the propofol group was lower than in the etomidate (Table 3). Those who received propofol needed less alfentanil than the etomidate group but the difference was not significant (Table 3).

The total incidence of side effects during maintenance (Table 4) was greater in the patients who received etomidate. One patient in the propofol group had bradycardia (below 50 beats/minute) after a subsequent dose of suxamethonium. This was easily treated with atropine 0.5 mg. Jet ventilation was performed in all cases without any problems.

The overall assessment of anaesthesia (Table 5) during maintenance was considered good in 12 patients in the propofol group and in five patients in the etomidate group. In seven of the latter patients and three of the former, the level of anaesthesia varied but was controlled easily. Coughing and movements indicated that an-

Table 4. Side effects during maintenance for both types of anaesthesia.

	Propofol ($n = 15$)	Etomidate ($n = 15$)	
Cough	2	8	$p < 0.02$*
Myoclonus	0	4	$p < 0.05$
Hypertension	0	3	NS
Bradycardia	1	0	NS
Hiccough	0	1	NS

* Fisher's exact test. NS, Not significant.

Table 5. Assessment of control and stability of anaesthesia.

	Propofol ($n = 15$)	Etomidate ($n = 15$)
Good	12	5
Adequate	3	7
Poor	0	3

Chi squared test. $p = 0.02$.

aesthesia was controlled inadequately in three patients in the etomidate group (Table 5). No patient complained of awareness.

Recovery from anaesthesia was rapid in both groups as indicated by the time from stopping the infusions until opening eyes on command. The patients who received etomidate had a tendency to take more time before they could respond correctly to simple questions (Table 2). During the first 10 minutes after the moment they opened their eyes on command, recovery of the patients given propofol was more complete as compared with the other group, as indicated by the Steward score (Table 6). After etomidate anaesthesia patients tended to drift back to sleep if undisturbed. One patient in the etomidate group was still asleep but rousable 15 minutes after opening his eyes on command.

The incidence of side effects during early and late recovery was higher following etomidate. One patient who received propofol complained of bad dreams during anaesthesia (Table 7). The high incidence of cough postoperatively was considered to be due to irritation of the upper airways by microlaryngeal surgery.

There was a discrepancy between the groups as concerns changes in blood pressure. After an initial decrease during induction with etomidate, blood pressure increased above pre-operative levels. The same pattern was seen with heart rate. In the propofol group, blood pressure decreased after induction and remained approximately 10% below pre-operative levels during the maintenance period. The heart rate was above pre-operative levels, but never significantly different. The change in heart rate was similar to that observed in the etomidate group (Fig. 1).

Discussion

Stable anaesthesia remaining at a deep level and rapid, uneventful recovery are the most important aims in microlaryngeal surgery.[2–4] Propofol and etomidate are short-acting intravenous agents whose half-lives are of the same order.[14,15] Recovery after infusion is rapid for both drugs. Therefore they both seem useful for microlaryngeal procedures. In this study the propofol group demonstrated better surgical conditions, more stable anaesthesia and, despite the same recovery times in both groups under investigation, better Steward recovery scores than the etomidate group. The question as to whether the dose regimens for the two drugs used in this study are fully comparable, is difficult to answer. For propofol we used an induction dose and infusion rates based on our experience in previous studies.[6] For etomidate we used the induction dose and infusion rates recommended by Fragen *et al.*[15] Higher doses of etomidate may result in better surgical conditions, but will also result in prolonged recovery.[4,15] It has been observed that during induction, propofol provides a better suppression of laryngeal reflexes than thiopentone.[16]

The generally accepted induction dose of propofol is 2.5 mg/kg, which results in induction times from 30 to 40 seconds.[6,10,11] A smaller induction dose of 2 mg/kg induced anaesthesia in only 80% of unpremedicated patients.[11] Robinson *et al.*[12] found a negative correlation between age and induction dose requirements. In this study we used an induction dose of 2 mg/kg preceded by a small dose of alfentanil; this resulted in successful induction of anaesthesia in

Table 6. Steward recovery scores (maximum score 6). Scores expressed as mean (range).

Time after opening eyes on command	Propofol (n = 15)	Etomidate (n = 15)	
3 minutes	5.5 (4–6)	4.5 (3–6)	p < 0.005*
5 minutes	5.9 (5–6)	5.1 (3–6)	p < 0.005
15 minutes	6.0 (6–6)	5.9 (4–6)	NS

* Analysis of variance. NS, Not significant.

Table 7. Side effects during recovery.

	Early			Late		
	Propofol (n = 15)	Etomidate (n = 15)		Propofol (n = 15)	Etomidate (n = 15)	
Cough	6	10	p < 0.1	2	6	p < 0.09*
Shivering	2	8	p < 0.02	0	0	NS
Vomiting	0	1	NS	0	1	NS
Nausea	0	3	NS	0	2	NS
Headache	0	1	NS	0	1	NS
Depression	0	2	NS	0	1	NS
Restlessness	0	2	NS	0	0	NS
Bad dreams	1	0	NS	0	0	NS

Early recovery is defined as the first 3 hours postoperatively, and late recovery from 3 up to 24 hours.

* Fisher's exact test. NS, Not significant.

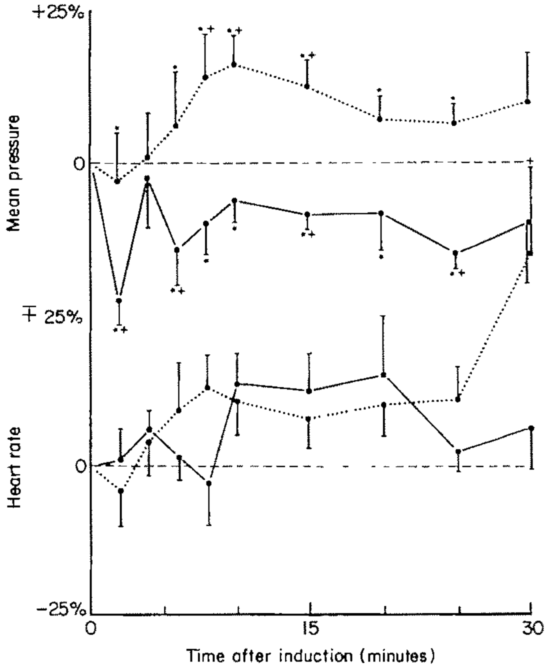

Fig. 1. Changes in mean arterial pressure and heart rate in relation to pre-operative values. Standard error of the mean is indicated by vertical bars. ——, Propofol; – – –, etomidate. * $p < 0.05$ between groups; $^+$ $p < 0.05$ in relation to pre-operative values.

all patients. Two factors contributed to this result: the rapid onset of action of alfentanil, and the relatively greater age of our patients. For both groups the low incidence of pain on injection was probably due to deliberate placement of the intravenous cannula in a large vein and the prior injection of a rapidly acting narcotic. This confirms the observations of Kay *et al.*[7] The changes in blood pressure in the propofol group were comparable with those reported by others.[7–9] The increase of blood pressure during etomidate anaesthesia may have been the result of too low an infusion rate of etomidate to guarantee an adequate level of anaesthesia for these procedures.

It is well known that intermittent bolus doses of suxamethonium for muscle relaxation may lead to severe bradycardia. Therefore atropine was added to the premedication. The reason that this technique has been chosen for muscle relaxation is the unpredictable duration of the procedure.

Prolonged adrenal suppression after etomidate administration has been demonstrated in both *in vitro* and *in vivo* studies.[17–19] In contrast, propofol has not been shown to interfere with steroid metabolism.[17] Therefore it is to be expected that the clinical use of propofol should not be accompanied by the same sort of problems as seen with etomidate.

In conclusion, propofol is preferable to etomidate as a hypnotic agent for total intravenous anaesthesia during microlaryngeal surgery with jet ventilation. More stable anaesthesia and better recovery are the main reasons. Adrenal suppression after etomidate administration may be another reason.

Acknowledgments

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Anaesthesia, 1987, Volume 42, pages 373–376

A comparison of propofol and methohexitone as induction agents for day case isoflurane anaesthesia

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Summary

The induction and recovery characteristics of equivalent doses of propofol and methohexitone were compared in 50 patients undergoing day case isoflurane anaesthesia. Propofol induction was smoother but was associated with greater cardiorespiratory depression. Both the speed and quality of recovery were superior with propofol compared with methohexitone.

Key words

Anaesthetics, intravenous; propofol, methohexitone.
Anaesthesia; outpatient.

Rapid recovery is a desirable feature of general anaesthesia for outpatients. Of the available induction agents, methohexitone results in quicker recovery than thiopentone and, in a recent study, MacKenzie and Grant[1] found that recovery after propofol was superior to that after methohexitone when used for induction in outpatient enflurane anaesthesia. Since this latter observation is at variance with that of previous workers,[2,3] the present study was undertaken to examine this in more detail. The present study compares induction and recovery in two series of patients induced with equivalent doses of either propofol or methohexitone. Maintenance was with nitrous oxide, oxygen and isoflurane, a new volatile agent which is rapidly eliminated. The study was approved by the local medical ethical research committee and patients gave verbal consent to participation.

Methods

Fifty healthy, unpremedicated patients aged 16–55 years, who weighed 45–80 kg and were to undergo minor gynaecological procedures, were randomly allocated to receive one or other induction agent. An induction dose of propofol 2.5 mg/kg or methohexitone 1.5 mg/kg was injected into a peripheral vein over 20 seconds. Maintenance was with 66% nitrous oxide in oxygen with an inspired concentration of up to 3% isoflurane which was reduced to 1–1.5% when adequate depth of anaesthesia was achieved.

Vital signs were monitored before and at 2-minute intervals during anaesthesia and note was made of complications such as pain on injection and induction side effects. The quality of induction was assessed subjectively as good, average or poor for each patient.

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At the end of the operation the patient was seen in the recovery ward by a second anaesthetist who was unaware of the induction agent used, and recovery was assessed using both clinical criteria and psychomotor methods. The former included the time to eye opening on command, orientation in time and place and giving of correct date of birth. Alertness was assessed on a five-point score,[4] and gait was assessed by straight-line walking and Romberg's test carried out at 20, 40 and 60 minutes after recovery. The four-choice reaction time test (CRT) was employed to measure psychomotor performance at 20, 40 and 60 minutes postoperatively and compared with pre-operative values. For the CRT measurement the subject studies a panel of four buttons and four lights which illuminate on a random basis. Immediately a light illuminates the subject presses the appropriate button to extinguish it. The response is recorded on magnetic tape and subsequently decoded and analysed by computer.[5] The latency of this response is an accurate measure of psychomotor performance.[6] An identical test was undertaken in a control group ($n = 25$) of gynaecological inpatients who had not yet undergone anaesthesia.

Postoperative side effects such as nausea, vomiting and headache were recorded. Results were analysed statistically using the Mann–Whitney U test, independent t-test, analysis of variance, Chi squared and Fisher's exact probability test.

Table 1. Patient data and duration of anaesthesia. Values expressed as mean (SEM).

	Propofol ($n = 25$)	Methohexitone ($n = 25$)	Control ($n = 25$)
Age, years	31 (1.8)	33 (2.1)	29 (1.5)
Weight, kg	57 (1.8)	59 (1.5)	60 (1.7)
Duration of anaesthesia, minutes	13 (1.2)	15 (1.2)	—

Results

The two groups were comparable with respect to age, weight and duration of anaesthesia (Table 1).

Induction characteristics. Table 2 summarises the observations made during induction of anaesthesia. Subjective side effects were more common with methohexitone than propofol, but only in the case of hiccough did this reach statistical significance ($p < 0.01$). The percentage decrease in systolic blood pressure was significantly greater ($p < 0.01$) after propofol than after methohexitone but the latter drug increased the heart rate to a greater degree than propofol. A short period of apnoea was a common finding with both drugs but this was statistically greater ($p < 0.05$) after propofol than following methohexitone.

Taking all these points into consideration, the induction was graded as good in 20 patients with propofol but in only 10 with methohexitone, whereas in only one patient with propofol was it considered to be poor compared with seven for methohexitone. The difference in this distribution is significant at the 1% level.

Recovery characteristics. Table 3 shows the mean recovery times as judged by clinical tests.

Table 2. Induction quality and side effects.

	Propofol ($n = 25$)	Methohexitone ($n = 25$)
Subjective side effects		
Cough	3	8
Hiccough	0	6
Laryngospasm	0	3
Excitatory	2	6
Pain on injection	12	14
Changes in vital signs		
Mean (SEM) percentage decrease in systolic blood pressure	12.1 (1.6)	1.0 (2.8)
Mean (SEM) increase in heart rate, beats/minute	7.1 (1.9)	20.0 (3.7)
Apnoeic period:		
0—30 seconds	10	16
30—60 seconds	10	9
>60 seconds	5	0

Table 3. Clinical recovery times. Values expressed as mean (SEM).

Mean time in minutes to:	Propofol	Methohexitone
Open eyes	7.9 (0.4)	11.1 (0.7)
Answer date of birth	8.3 (0.5)	11.7 (0.7)
Orientation in time and place	8.3 (0.5)	11.6 (0.7)

Recovery was significantly more rapid ($p < 0.01$) with propofol using all three endpoints.

With all three endpoints recovery was significantly more rapid ($p < 0.01$) with propofol than after methohexitone. At 20 minutes postoperatively ataxia was common in both groups, although patients who had received propofol were more alert ($p < 0.01$) than those who had been given methohexitone (Fig. 1). By 40 minutes only one patient showed ataxia following propofol as compared with 10 after methohexitone, and this is a highly significant difference ($p < 0.01$) between the two groups (Table 4).

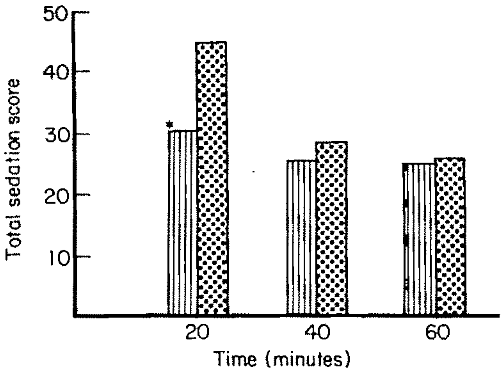

Fig. 1. Total sedation scores at 20, 40 and 60 minutes postoperatively. ▥, Propofol; ▦, methohexitone. * Significant at $p < 0.01$.

Table 4. Frequency of postoperative sequelae.

	Propofol	Methohexitone
Ataxia		
20 minutes	16	17
40 minutes*	1	10
60 minutes	0	1
Headache	3	6
Emetic symptoms	2	5

* Significant difference ($p < 0.01$) between the drug groups.

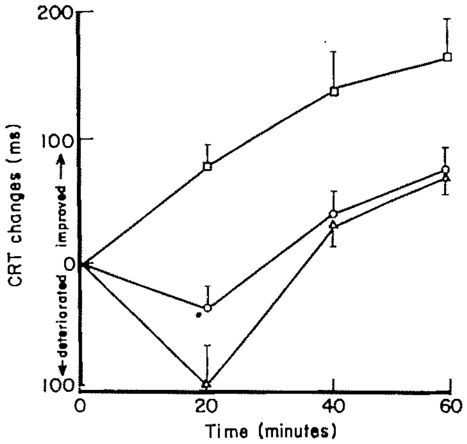

Fig. 2. Changes from baseline CRT values (mean, SEM). □——□, control; ○——○, propofol; Δ——Δ, methohexitone. * Significant at $p < 0.05$.

Figure 2 summarises the choice reaction times, which were significantly better in the propofol group at 20 minutes compared with methohexitone, but by 40 minutes there was no difference between the two series. Likewise, there were no significant differences in minor postoperative sequelae between the two groups (Table 4).

Discussion

This paper shows some similarities and some differences between the two agents used for induction of anaesthesia in outpatients. Both are rapidly acting compounds and pain on injection was equally common in both series. This can be minimised by the use of large forearm veins and it is not a problem in clinical practice.[7] While subjective side effects were less with propofol, the new agent did produce greater cardiovascular and respiratory depression than methohexitone. The smaller decrease in systolic blood pressure and the greater increase in heart rate seen after methohexitone may be explained in part by the higher incidence of 'turbulent' inductions with this drug. The differences as regards hypotension were not considered to have any clinical importance by the anaesthetists involved and apnoea, although more common with propofol, does not cause a major problem in anaesthetic practice. Judging by both subjective observations and psychomotor testing, recovery was more rapid with propofol than methohexitone and this is important when early ambulation is desired.

In conclusion, propofol is preferred to

methohexitone for induction of anaesthesia in outpatients because of the fewer induction complications and more rapid recovery with the new drug.

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The loading dose for continuous infusion epidural analgesia

A technique to reduce the incidence of hypotension

D. M. MACLEOD, H. K. TEY, G. F. BYERS, W. C. DOLLERY AND M. E. TUNSTALL

Summary

The effects of 0.08% and 0.5% bupivacaine, given in equal volumes as the loading dose prior to continuous infusion epidural analgesia, were compared. The incidence of hypotension, as well as the height of sensory blockade, was markedly lowered by reducing the loading dose of bupivacaine. However, the efficacy of the epidural analgesia was maintained by using the same volume for the loading dose. The mean onset of analgesia was delayed from 9.1 to 13.6 minutes. There were fewer calls for intervention consequent upon the reduction in adverse effects. The use of 8 ml 0.08% bupivacaine for the loading dose or for subsequent top-ups is recommended. The concept of a liquid sleeve of local anaesthetic in the epidural space is further supported.

Key words

Anaesthetic techniques, regional; epidural.
Anaesthetics, local; bupivacaine.

A recent study at this hospital compared two concentrations of bupivacaine for continuous epidural infusion.[1] There was a relatively high incidence of hypotension but nearly all episodes were related to the loading (initial bolus) dose of local anaesthetic. For intermittent bolus epidural analgesia, top-ups are given when pain recurs. In continuous infusion epidural analgesia, an infusion of bupivacaine is started without waiting for the effect of the bolus doses to wear off. If an equal amount of bupivacaine is used to initiate both techniques, more adverse effects would be expected if an infusion is also given.

A patchy block may result if the volume of the loading dose is reduced.[2] An unacceptable delay in the onset of analgesia may result if the infusion of bupivacaine is commenced immediately after a test dose, but without a loading dose. In the experience of one of us (D.M.M.), the regular use of 0.08% bupivacaine as a loading dose to initiate epidural analgesia appeared to be very satisfactory. It was decided to compare this technique with that used by the other anaesthetists in Aberdeen where a loading dose of 0.5% bupivacaine was customary.

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Methods

Patient selection

The subjects, from a consecutive series of patients who required epidural analgesia for labour pain, were randomly allocated to two groups. Patients with cardiac disease, recent antepartum haemorrhage, pre-eclamptic toxaemia under chlormethiazole infusion or who required resiting of their epidurals, were not studied.

Both groups were given a 3-ml 0.5% bupivacaine test dose, followed by the loading dose; group A received 8 ml 0.08% bupivacaine and group B, 8 ml 0.5% bupivacaine. The further conduct of the epidural analgesia was similar except for the subsequent top-ups.

Technique

An intravenous infusion was established. A Portex epidural catheter was placed 4 cm into the epidural space, using a 16-gauge Tuohy needle inserted at the L_3–L_4 interspace. Analgesia was induced in the left lateral decubitus position. A test dose of 3 ml bupivacaine 0.5% was injected via a Millipore filter after negative aspiration for blood or cerebrospinal fluid. The loading dose was given once it had been confirmed that there were no untoward sequelae from the test dose. Bupivacaine 0.08% at 25 ml/hour via a volumetric infusion pump was commenced at the time of the loading dose or as soon as possible thereafter. This solution was made up by adding 48 ml of 0.5% bupivacaine to a 250-ml bag of normal saline. Bolus top-ups of 4–8 ml 0.08% bupivacaine in group A or 0.5% bupivacaine in group B were used if required.

Observations

The arterial blood pressure was recorded before the epidural and every minute for the first 5 minutes after the loading dose, then at 5-minute intervals for 30 minutes, and subsequently at 45 minutes, 60 minutes, and thereafter half-hourly until the epidural infusion was discontinued. Measurements below 100 mmHg were recorded as episodes of hypotension. All blood pressure recordings were performed by midwives who were not aware of the allocated group.

The height of sensory block (loss of temperature sensation to ice), the degree of motor block (0, no leg weakness; 1, able to lift the foot clear of the bed; 2, able to slide the foot up the bed; 3, unable to slide the foot up the bed), and the site and degree of any pain sensation were all noted. Recordings were taken at 15, 30, 45, 60 and 120 minutes, and thereafter 2-hourly.

If the anaesthetist was called to see the patient or adjusted the set technique when assessing subjects, this was recorded as an intervention. The mothers were interviewed about the pain relief and leg weakness by a different anaesthetist on the following day.

Analysis of results

Student's *t*-test for parametric data and Fisher's exact test for nonparametric data were used.

Results

There were 30 subjects in group A (0.08% bupivacaine) and 31 in group B (0.5% bupivacaine). The loading dose was given within 11 minutes after the test dose except for one dose in each group (within 25 minutes). There were no significant differences between the two groups with respect to parity, gestation, age, height, weight, pre-epidural systolic blood pressure and duration of epidural.

Significantly fewer patients in group A had hypotension than in group B (Table 1). Except for one patient in group A (two episodes) and two patients in group B (one episode each), all the hypotensive episodes occurred within 30 minutes after the loading dose. Subsequent top-ups for pain relief using 4–8 ml of either concentration did not cause hypotension.

The mean onset of satisfactory analgesia was significantly slower in group A (13.6 minutes) than in group B (9.1 minutes), as shown in Table 2. During the first hour after the loading dose, analgesia remained satisfactory in all cases except for one in group B.

The intensity of pain episodes as recalled at

Table 1. Number of patients with hypotension.

Group	Without hypotension	With hypotension
A	28	2
B	20	11

Significant difference between groups ($p < 0.05$)

the postdelivery interview (Table 3) and the incidence of pain that required intervention (Table 4) were similar, with few differences between groups A and B.

Table 2. Time to onset of satisfactory analgesia: mean (SEM).

Group	Time to onset of satisfactory analgesia, minutes*
A	13.6 (1.8)
B	9.1 (0.9)

* Significant difference between groups ($p < 0.05$). Data exclude one patient in group A and two in group B with satisfactory analgesia before the loading dose.

Table 3. Postdelivery interview: pain assessment (number of mothers assessing worst pain episode during epidural).

Group	No pain	Slight	Bearable	Unbearable
A	11	9	7	3
B	12	9	7	3

There was no significant difference in the maximum degree of leg weakness attained nor in the time to reach it (Table 7).

In group A, 0.08% bupivacaine was used for top-ups when pain recurred. However, in two patients, 5–8 ml 0.5% bupivacaine was also used when 0.08% bupivacaine was ineffective. Despite this, complete analgesia was not achieved in either patient. In group B, 4–8 ml 0.5% bupivacaine was used.

The total bolus dose (mg) given during the epidural was significantly lower in group A (Table 8) but there was no statistical difference in the total amount and overall rate at which bupivacaine was administered. There were more stoppages and reduced rates of infusion in group B, due to the problems shown in Table 4.

More patients required interventions in group B as a result of a significantly higher incidence of high blocks and hypotensive episodes (Table 4). Five out of 31 in group B had marked interscapular pain but none in group A. However, this was not statistically significant. In both

Table 4. Analysis of number of patients who required intervention.

Group	Hypotension*	High block*	Recurring pain	Delayed onset of satisfactory analgesia	Interscapular pain
A	2	5	8	2	0
B	11	14	8	0	5

* Significant difference between groups ($p < 0.05$).

Table 5. Time to reach the highest level of block on either side.

Group	Mean (SEM) time to highest level of block, minutes*	Number of subjects who attained highest level*	
		Within 1 hour†	After 1 hour
A	143.0 (23.1)	13	17
B	44.0 (5.5)	27	4

* Significant difference between groups ($p < 0.01$).
† Includes two patients in group A and three in group B with infusion times less than or equal to 60 minutes.

The time to reach the highest level of block on either side was significantly less in group B. Eighty-seven percent reached it within 1 hour after the loading dose, in contrast to only 43% in group A (Table 5). The number of subjects with sensory block above T_5 on either side was significantly greater in group B, as shown in Table 6, which also displays the mode and range.

Table 6. Highest level of sensory block attained on either side.

Group	Thoracic level*		Mode	Range
	1–4	5–12		
A	8	22	T7	T3–T10
B	18	13	T4	T2–T8

* Significant difference between groups ($p < 0.05$).

Table 7. Maximum degree of leg weakness.

Group	Mean (SEM) time to maximum weakness, minutes*	Number of subjects who attained maximum degree of weakness	
		Grades 0–1	Grades 2–3
A	156.5 (26.2)	7	23
B	110.4 (27.3)	8	23

No significant differences between groups.
* Excludes two patients in each group with no leg weakness at all.

Table 8. Amount of bupivacaine administered: mean (SEM).

Group	Total bolus doses, mg*	Total infusion dose, mg	Total dose, mg	Overall rate of administration, mg/hour
A	27.8 (3.3)	125.2 (12.5)	153.0 (14.4)	25.3 (1.5)
B	63.1 (2.8)	110.2 (13.7)	173.2 (14.7)	29.4 (2.4)

* Significant diference between groups ($p < 0.01$).

groups, eight patients required interventions for pain. Two in group A needed an additional 8 ml 0.08% bupivacaine for delayed onset of analgesia. There were no differences in the mode of delivery between the groups.

Discussion

The continuous infusion of local anaesthetic into the epidural space has many advantages over intermittent bolus top-ups.[1,3–10] Epidural infusions to maintain analgesia offer increased safety from complications that result from intrathecal or intravascular migration of the epidural catheter.[7] There is less risk of contamination within a closed system.[8] In hospitals where midwives do not administer the epidural top-ups, there can be delays that result in more pain and less satisfactory analgesia. This method allows the anaesthetist to visit the patient when he is free rather than being summoned the moment the pain returns. This, of course, does not diminish his responsibility for the epidural.[5,8,10]

Continuous infusion epidural analgesia has been used widely for the epidural service in Aberdeen during the last 3 years and is considered to be most satisfactory by the patients, the anaesthetists and the midwives. One criticism of continuous epidural infusion of bupivacaine at 20 mg/hour is the high incidence of hypotension.[1] However, the primary cause is not the continuous infusion, but the initial loading dose. It should be noted that only three patients out of 61 in our study developed hypotension later than 30 minutes after the loading dose. None of these had a systolic blood pressure below 90 mmHg.

This study shows a marked reduction in the incidence of hypotension if the loading dose is reduced but the same volume retained Preloading with intravenous fluids was not used. The epidural analgesia was effective with only a minimal delay in the onset of analgesia. If satisfactory analgesia is not attained within 20 minutes, a further dose of 8 ml 0.08% bupivacaine should be given. This was effective, with no adverse effects, in the two cases shown in Table 4. The reduction in the loading dose also resulted in lower levels of sensory blockade: significantly fewer epidural blocks reached T_4.

It is interesting that the overall amount and rate of bupivacaine administered were similar in both groups yet there were more side effects in group B. This suggests that the loading dose plays a prominent role in the subsequent course of the continuous epidural infusion. Possibly the loading dose already in the epidural space is pushed cephalad by the continuous infusion. In group B, this could result in the concentrated solution affecting more thoracic segments. Perhaps this also explains the high incidence of hypotension during the early stages of the techniques used by Ewen *et al.*[1]

In group A, the test dose was 15 mg bupivacaine but the loading dose was only 6.4 mg. It is unusual, though not rare, for a 15-mg test dose to give adequate analgesia for labour pain.

The effect of 8 ml for the loading dose in group A is to spread out the 15 mg bupivacaine previously administered from the test dose. A patchy block achieved merely by reduction in the volume of the loading dose is thus avoided. This would confirm the concept of a liquid sleeve that maintains local anaesthetic activity in the epidural space.[1,11]

The technique, using 8 ml 0.08% bupivacaine as a loading dose, improves the safety of continuous infusion epidural analgesia without compromising the quality of analgesia and calls for fewer interventions in the conduct of the epidural.

Acknowledgments

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Anaesthesia, 1987, Volume 42, pages 382–386

The effect of sufentanil on the cardiovascular responses to tracheal intubation

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Summary

The effects of sufentanil 0.5 or 1 μg/kg, given intravenously after induction of anaesthesia, on the cardiovascular responses to tracheal intubation were examined in a controlled, randomised, double-blind investigation. The control group of patients exhibited significant rises in arterial blood pressure and heart rate for 4 minutes after tracheal intubation. Heart rate exceeded 100 beats/minute and systolic pressure increased by over 20% in every patient. All patients moved or breathed within 10 minutes of the administration of suxamethonium. Sufentanil 0.5 μg/kg prevented increases in the mean values of heart rate and arterial blood pressure, although increases were observed in five patients. Significant falls in the mean values of heart rate and arterial pressure occurred from 4 minutes after intubation until observations ended 15 minutes after induction of anaesthesia. Two patients moved or breathed during this time, although movement in response to nerve stimulation occurred in all patients 10 minutes after administration of suxamethonium. Sufentanil 1 μg/kg was effective in suppressing a rise in heart rate or arterial pressure in every patient. Significant falls in these variables occurred from 2 minutes after tracheal intubation onwards. No patient moved or breathed for 15 minutes after induction of anaesthesia, although neuromuscular transmission was present 10 minutes after giving suxamethonium in each case.

Key words

Intubation, tracheal; complications.
Analgesics, narcotic; sufentanil.

Sufentanil is the most potent opioid available for use in man. It has been shown to be useful as the analgesic component of general anaesthesia, with a similar effect, speed of onset and duration of action after a single intravenous injection to ten times the dose of fentanyl.[1] However, sufentanil has a shorter elimination half-life than fentanyl and is probably less cumulative. This results in a potency ratio of approximately 1:7 when the drugs are given by repeated injections.[1,2] Sufentanil is the least toxic opioid in dogs whose lungs are mechanically ventilated[3] and allows better cardiovascular control than fentanyl during neurosurgical anaesthesia.[4]

Laryngoscopy and tracheal intubation after the use of an intravenous anaesthetic and suxamethonium, local anaesthesia or light general anaesthesia have frequently been shown to cause cardiac dysrhythmias.[5–7] Such cardiovascular

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disturbances may be dangerous in some patients, for instance those suffering from myocardial disease.

The cardiovascular responses to tracheal intubation may be limited by the administration of some opioids at induction of anaesthesia. Fentanyl 5 μg/kg and alfentanil 30 μg/kg have been shown to be effective,[5] whereas morphine 0.59 mg/kg and pethidine 4.34 mg/kg were not; indeed, this dose of pethidine given alone caused a significant tachycardia.[2] In view of its potency and cardiovascular stabilising effects, a study was undertaken to determine whether intravenous sufentanil in doses of 0.5 or 1 μg/kg was effective in controlling the cardiovascular responses to laryngoscopy and tracheal intubation after induction of anaesthesia using thiopentone and suxamethonium.

Method

The investigation was approved by the hospital ethical committee, and was controlled and double-blind. Thirty patients, ASA class 1 or 2, between 18 and 60 years old who were to undergo surgery that required tracheal intubation and who had given written informed consent to participate in the study, were allocated randomly into three groups of ten. They received at induction of anaesthesia either saline, sufentanil 0.5 μg/kg or sufentanil 1 μg/kg. The test medications used were prepared so as to be identical in appearance, in a volume of 10 ml, by an anaesthetist otherwise unconnected with the study; they were given to the unpremedicated patients over 30 seconds immediately after induction of anaesthesia by thiopentone 4 mg/kg also given over 30 seconds. Suxamethonium 1.5 mg/kg was then injected as rapidly as possible and laryngoscopy and tracheal intubation carried out after a further 90 seconds. Ventilation to normocapnia was maintained using nitrous oxide 70% in oxygen and monitored using the Datex capnograph.

Monitoring was started before induction of anaesthesia. Arterial blood pressure and heart rate were recorded automatically each minute using the Dinamap recorder and the ECG (CM5 lead) was displayed continuously. The mean of the three observations immediately preceding induction of anaesthesia was taken as the control value. Observations were continued until 15 minutes after induction or until the patient breathed spontaneously or moved; this time was noted. Anaesthesia appropriate to the surgery being performed was then immediately instituted. Ten minutes after the administration of suxamethonium, the recovery of neuromuscular transmission was confirmed by nerve stimulation in those patients who had not exhibited somatic responses. Statistical analysis was by Student's paired *t*-test.

Results

Figures 1 and 2 illustrate the changes in heart rate and systolic arterial blood pressure in the three groups. The patients who received saline showed a significant increase in heart rate, systolic and diastolic arterial pressure for 4 minutes after tracheal intubation. In every patient heart rate exceeded 100 beats/minute and systolic pressure increased by more than 20%. One patient breathed spontaneously 4 minutes after tracheal intubation; by 5 minutes four patients and by 6 minutes seven patients moved or breathed, and all ten patients responded by 10 minutes after intubation. The time for recovery of breathing or movement in 50% of the patients (obtained by interpolation) was approximately 5.5 minutes after intubation. Three patients developed extrasystoles (two ventricular).

There was no rise in mean systolic or diastolic arterial pressure or heart rate in the patients given sufentanil 1 μg/kg; indeed, in none of these patients was a rise in heart rate observed at any observation time after intubation, nor were any dysrhythmias observed. One minute after intubation there were no significant changes in the mean values of the variables, but thereafter there was a significant reduction in arterial pressures and heart rate throughout the observation period ($p < 0.05$, paired *t*-test). No patient breathed or moved for 15 minutes following induction of anaesthesia, although muscle response was observed in all patients 10 minutes after administration of suxamethonium.

No rise in the mean values of systolic and diastolic arterial pressure or heart rate occurred following administration of sufentanil 0.5 μg/kg. No significant changes were observed for 3 minutes after tracheal intubation, but from 4 minutes onwards significant slowing of heart rate and reduction in arterial pressure were observed. Small increases in heart rate and arterial pressure were observed in five of the

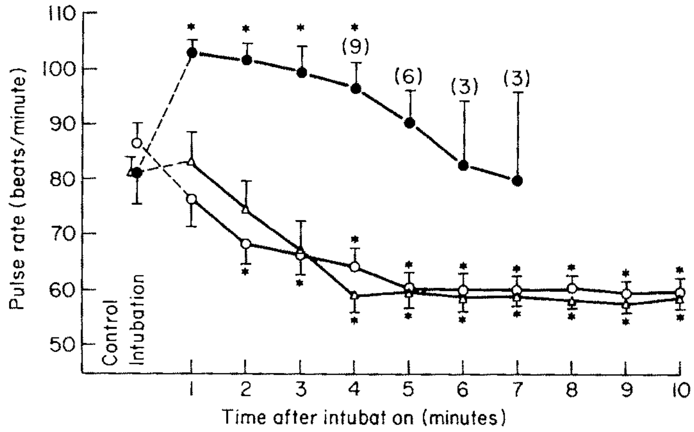

Fig. 1. Changes in pulse rate following tracheal intubation. The mean and SEM changes in pulse rate following tracheal intubation are displayed for three groups of ten patients, who received intravenous saline (●), sufentanil 0.5 μg/kg (△) or sufentanil 1 μg/kg (○) after induction of anaesthesia. $^*p < 0.01$ compared with control (pre-anaesthetic) values (paired *t*-test). Figures in parentheses indicate μ when less than 10.

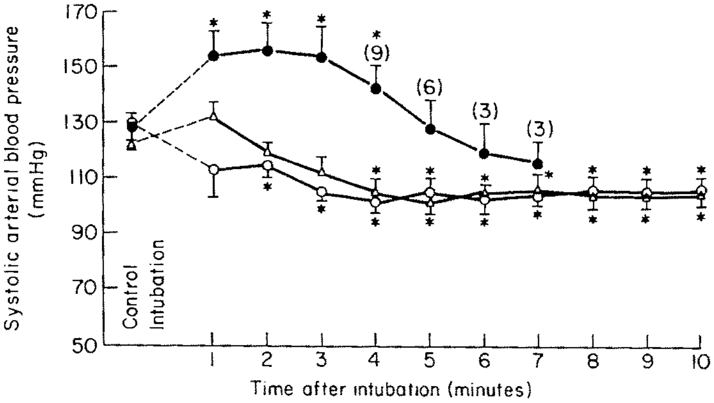

Fig. 2. Changes in systolic arterial blood pressure following tracheal intubation. The mean and SEM changes in systolic arterial blood pressure following tracheal intubation are displayed for three groups of ten patients, who received intravenous saline (●), sufentanil 0.5 μg/kg (△) or sufentanil 1 μg/kg (○) after induction of anaesthesia. $^*p < 0.01$ compared with control (pre-anaesthetic) values (paired *t*-test). Figures in parentheses indicate μ when less than 10.

patients following intubation. No dysrhythmias were seen. Spontaneous breathing and movement were seen in two patients in this group at 4 and 5 minutes after intubation. Despite the return of neuromuscular transmission 10 minutes after giving suxamethonium, no other patients exhibited a somatic response during the observation period. No complications or side effects were observed in the patients studied.

Discussion

The results observed here correspond closely with those reported in earlier studies.[5,8,9] The intensity and duration of cardiovascular responses to tracheal intubation in the control group who received saline were similar to those recorded in the three earlier investigations, which were conducted using the same anaesthetic technique, study programme and similar monitoring.

The effects of sufentanil 0.5 μg/kg were similar to those observed by Black *et al.*[5] and Kay *et al.*[8] following administration of fentanyl 5 μg/kg. No increases in mean heart rate or arterial pressure occurred following intubation, but small increases were observed in individual patients both after sufentanil 0.5 μg/kg and after fentanyl 5 μg/kg. Significant decreases in mean heart rate and arterial pressure commenced 4 minutes after intubation both in the patients who received sufentanil 0.5 μg/kg and in those who received fentanyl 5 μg/kg. No abnormalities of cardiac rhythm were observed in any of these groups. Two patients moved during the observation period after administration of sufentanil 0.5 μg/kg, compared with one or two (twice) in the three groups of patients who received fentanyl 5 μg/kg. The results correlate with previous experience which indicates a relative potency of 1:10 for the two opioids.[1]

Sufentanil 1 μg/kg had a demonstrably greater effect than sufentanil 0.5 μg/kg, and was sufficient to suppress completely all cardiovascular responses to intubation in all the patients observed. Significant decreases in mean heart rate and arterial pressure were observed 2 minutes after intubation. These occurred sooner than after the smaller dose and no patient moved or breathed during the observation period, despite the demonstrated absence of neuromuscular block 10 minutes after suxamethonium.

The onset of effect was faster after sufentanil 1 μg/kg than after sufentanil 0.5 μg/kg, but the eventual effect of these two doses of the drug on heart rate, systolic and diastolic arterial pressure was the same: the larger dose does not slow the heart or depress blood pressure more than the smaller dose. This impression of the cardiovascular stability induced by sufentanil is reinforced by the small degree of variation in cardiovascular parameters seen between and within patients once the full effects of the drug are observed.

The investigation supports the view that it is appropriate to give sufentanil following induction of anaesthesia and immediately before the administration of suxamethonium, thereby avoiding the problems of the stiff-chest syndrome sometimes observed after intravenous opioids. If these problems are discounted, it may be that the earlier administration of sufentanil 0.5 μg/kg would completely prevent all cardiovascular responses in every individual, since the results of this investigation suggest that the full effect of the opioid was not apparent at the time of maximum stimulation. However, in an earlier study which compared the use of intravenous fentanyl 5 μg/kg given after and 5 minutes before induction of anaesthesia,[5] the average effects were similar in both groups and individuals whose heart rate or arterial pressure increased were observed in the group given fentanyl before induction.

Sufentanil given intravenously is, as are fentanyl and alfentanil, an effective drug for the control of the cardiovascular responses to tracheal intubation. The choice of sufentanil and the appropriate dose to use will depend on other factors. A dose of 1 μg/kg would appear to be appropriate if it is necessary to prevent any rise in cardiac rate or arterial pressure in a particular patient, but will cause earlier depression of these variables and probably more persistent depression of breathing than 0.5 μg/kg, which like fentanyl 5 μg/kg is likely to prevent clinically significant cardiovascular responses in the majority of patients. On the basis of comparability of effect with ten times the dose of fentanyl, sufentanil 1 μg/kg is likely to cause significant respiratory depression for about 45 minutes. When suppression of cardiovascular responses to tracheal intubation is required during anaesthesia of a shorter duration, the use of alfentanil 30 μg/kg would appear to be appropriate.[5]

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Vecuronium infusions in patients with renal failure in an ITU

C. L. SMITH, J. M. HUNTER AND R. S. JONES

Summary

The use of an infusion of vecuronium is described in seven patients with renal and respiratory failure in an intensive therapy unit. Neuromuscular function was monitored throughout using the train-of-four twitch technique. A bolus dose of vecuronium (0.1 mg/kg) was given, followed immediately by a continuous infusion (0.05 mg/kg/hour). The infusion rate was adjusted until the first twitch of the train was below 20% of control and then run at a constant rate.

There was a marked variation in the dose of vecuronium administered (0.01–0.65 mg/kg/hour). Two patients, who appeared to be most sensitive to the drug, were both receiving metronidazole. Recovery of neuromuscular function was extremely prolonged and widely variable (6–37 hours) on stopping the infusion. No adverse cardiovascular effects or evidence of histamine release were seen as a result of administration of the drug. Vecuronium is probably more suitable for administration in bolus doses rather than by infusion in patients with renal and respiratory failure.

Key words

Anaesthetic techniques; infusions.
Neuromuscular relaxants; vecuronium.

Patients with respiratory failure in the intensive therapy unit (ITU) who require intermittent positive pressure ventilation (IPPV) are routinely given sedative drugs such as benzodiazepines, and narcotic analgesics such as morphine. If the lung compliance is very poor, however, such central depressant drugs may be insufficient and muscle relaxants are then frequently brought into use. Conventional non-depolarising muscle relaxants, such as pancuronium and alcuronium, are usually given in bolus doses in such circumstances and, unless the patient has renal failure, these drugs provide satisfactory conditions for IPPV. As these drugs are excreted by the kidney, however, they may produce excessively prolonged neuromuscular blockade in the patient with no renal function. This should not be true of the newer competitive relaxants, vecuronium and atracurium, since renal excretion is unimportant and these agents would seem to be more suitable for this type of patient.

As both of the newer drugs have a shorter duration of action than other competitive relaxants it would also seem, at first sight, more appropriate to administer them by infusion, since bolus doses might be required too frequently

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to be practicable. Atracurium infusions have already been shown to be a useful adjunct in the patient with respiratory and renal failure who requires IPPV and dialysis in the ITU;[1] the particular benefit of the drug is the rapid recovery from neuromuscular blockade on stopping the infusion. This is of great practical benefit when it is planned to start weaning the patient from IPPV and also when it is necessary to assess cerebral status.

Little information is available on the use of vecuronium infusions in patients with multisystem organ failure in the ITU. Only one limited study has been reported and in this the drug was not given on a weight basis nor was the length of time during which it was administered reported. In addition, no information is available on the degree of renal dysfunction present in the patients described.[2] It was therefore decided to study the use of this drug in patients with respiratory and renal failure; these patients incidentally had varying degrees of hepatic dysfunction (usually as a result of septicaemia). Particular attention was paid to the recovery of neuromuscular function once the infusion of vecuronium had stopped.

This study was approved by the hospital ethical committee and permission was obtained from the patients' next of kin.

Methods

Seven patients were studied, although patient 6 died before the investigation was complete. All needed IPPV on admission to the ITU and all were in acute renal failure. Clinical details and the antibiotic therapy used in each patient are given in Table 1. Blood gas analysis, urea, electrolyte and liver function tests from each patient on admission are given in Tables 2 and 3. Each patient was established on an infusion of morphine (4–10 mg/hour) before the study commenced and was given intermittent doses of midazolam to ensure good sedation throughout the period of investigation.

Neuromuscular function was monitored throughout the study using the train-of-four twitch technique with mechanical recording methods described in detail elsewhere.[3] Control values were obtained once the equipment was set up. Six patients had already had a tracheal tube inserted either on admission to the ITU, or

Table 1. Presenting complaints of each of the patients studied, together with the complicating factors which precipitated respiratory and renal failure. The antibiotics used during the period of study are also given, as is the eventual outcome of treatment.

Patient	Age (years)	Presenting complaint	Complications (causative organism)	Antibiotic therapy	Outcome
1	47	Mitral valve replacement	Septicaemia (LFC), DIC	Cefotaxime	Died
2	16	Perforated duodenal ulcer	Peritoneal sepsis Septicaemia (*Candida albicans*) ARDS	Amphotericin Flucytosine Piperacillin Tobramycin	Survived
3	48	Acute haemolytic anaemia (drug-induced)	Haemoptysis Chest infection (LFC)	Piperacillin	Survived
4	47	Chronic renal dysfunction Chest infection	Peritoneal sepsis from dialysis cannula (*Klebsiella*)	Cefotaxime Erythromycin	Survived
5	57	Sigmoid volvulus Faecal peritonitis	Septicaemia ARDS	Cefotaxime Metronidazole	Survived
6	26	Viral pneumonia	ARDS	Erythromycin Metronidazole Tobramycin Cefotaxime	Died
7	65	Upper and middle lobectomy	Multiple pulmonary emboli ARDS	Erythromycin Metronidazole Tobramycin	Died

LFC, Lactose-fermenting coliforms; DIC, disseminated intravascular coagulation; ARDS, adult respiratory distress syndrome.

Table 2. Arterial blood gas analyses for each of the seven patients at the commencement of the vecuronium trial, together with ventilation mode, amount of end expiratory pressure used and the derived shunt value.

	Patient						
	1	2	3	4	5	6	7
$F\text{IO}_2$	0.6	0.45	0.6	0.6	0.6	0.65	0.5
pH	7.44	7.31	7.39	7.39	7.36	7.29	7.40
$P\text{CO}_2$ (kPa)	5.4	4.6	4.65	5.47	4.78	4.78	4.76
$P\text{O}_2$ (kPa)	10.87	11.47	12.83	8.49	10.67	15.6	11.2
Base excess (mmol/litre)	3.0	−7.8	−3.0	−0.2	−4.5	−8.8	−1.9
Ventilation	CMV	CMV	CMV	CMV	CMV	CMV	CMV
PEEP (kPa)	0.77	0.75	1.0	0.5	0.5	0.5	0.5
Shunt	25%	15–20%	20%	25%	25%	20%	20%

CMV, Continuous mandatory ventilation; PEEP, positive end expiratory pressure.

Table 3. Urea, electrolyte and liver function tests on commencement of the trial for each of the seven patients.

	Patient						
	1	2	3	4	5	6	7
Sodium (130–150 mmol/litre)	143	131	134	131	135	141	130
Potassium (3.6–5.4 mmol/litre)	3.8	4.9	4.3	4.7	4.6	4.5	4.4
Urea (3–7 mmol/litre)	12.8	26.4	38.6	28.5	21.5	36.0	27.6
Creatinine (60–110 μmol/litre)	383	450	450	947	492	683	577
Total bilirubin (2–17 μmol/litre)	155	12	345	7	69	95	45
Direct bilirubin (1–6 μmol/litre)	104	0	275	3	57	78	28
Alanine transferase (7–45 U/litre)	4150	28	520	56	156	238	104
Glutamyl transferase (0–65 U/litre)	52	30	57	40	64	56	92
Alkaline phosphatase (35–130 U/litre)	82	146	119	177	135	160	197

sometime before the study commenced; in the other patient the tube was passed immediately after the first dose of relaxant had been given.

The initial dose of vecuronium (0.1 mg/kg) was given through a central line. Immediately after this an infusion of the drug was commenced into a peripheral vein which was used solely for this purpose, initially at the rate of 0.05 mg/kg/hour. The aim of the study was to keep the first twitch of the train just visible on the tracing and certainly less than 20% of control. To achieve this, the infusion rate was altered one hour after the beginning of the study by ±0.005 mg/kg/hour and this procedure was repeated at 30-minute intervals until the required conditions had been achieved for at least one hour. The infusion was then allowed to run at a fixed rate.

Once the study had commenced, monitoring lines (arterial and pulmonary artery catheters) and a dialysis line were inserted if this had not been done already, and the patient's cardiovascular condition stabilised as far as possible using inotropic agents if necessary. Five of the patients required inotropic support with dopamine during the study and patients 1, 6 and 7 required further inotropic support with both dobutamine and adrenaline infusions. Dialysis was then commenced. The infusion was discontinued when improved respiratory function, often effected by the removal of fluid during dialysis, made the further use of a muscle relaxant unnecessary.

Neuromuscular monitoring continued throughout the period of the vecuronium infusion, except during the hours from midnight to 07:00 hours when, if the height of the train-of-four was stable, continuous monitoring was stopped and the response to the train checked at 2-hourly intervals only. Neuromuscular monitoring was always re-established prior to the termination of the vecuronium infusion and continued throughout the period of recovery of neuromuscular function. It was stopped only when the latter was considered adequate. Satisfactory recovery was taken as the return of the first twitch of the train (A′) to 75% of control (A) and the return of the fourth twitch (D′) to 75% of A′, or when these two variables had remained constant for at least one hour.

Results

The initial bolus dose of vecuronium (0.1 mg/kg) ablated the train-of-four in six of the seven patients: in the other patient (3), A′/A decreased to 4% and D′/A′ disappeared completely. The period of adjustment of the infusion of vecuronium to produce satisfactory control of the train then took 1.25–5 hours.

In four of the seven patients, the train gradually disappeared over the next 0.75–3 hours after the main infusion rate had been fixed (Table 4). In the remaining two cases (4 and 5), the first twitch of the train was still present when the infusion was stopped.

The duration of each infusion and the total dose of vecuronium given are detailed in Table 4, together with the height of A′/A and D′/A′ when the infusion was stopped and at the completion of the study. The time from stopping the infusion until completion of the study is also given.

The mean infusion time was 20.5 hours (range 6.75–32.0 hours). There was a large variation in the mean dose of vecuronium required by infusion to produce satisfactory, but not excessive depression of the train-of-four: the dose used varied from 0.01 to 0.065 mg/kg/hour. This dose did not appear to be related to the degree of hepatic dysfunction present. Thus in patient 3, who had significant liver dysfunction (Table 3), a vecuronium infusion of 0.065 mg/kg/hour was needed to control the twitch response, whilst in patient 5, who had only slightly deranged liver function, a lower infusion rate sufficed (0.15 mg/kg/hour).

It was noted that in the two patients who received parenteral metronidazole during the study (cases 5 and 6), only a small dose of vecuronium was required. Thus, cases 5 and 6 received only 0.015 and 0.01 mg/kg/hour, respectively. Although case 7, who had recently completed a course of metronidazole, received a much larger dose of vecuronium than patients 5 and 6 (0.05 mg/kg/hour), recovery was prolonged markedly (see below). Two of these three patients who received metronidazole (cases 6 and 7), also received tobramycin during the period of the trial as did patient 2 who, in contrast, was not as sensitive to vecuronium.

The cardiovascular status of patient 6 deteriorated markedly while she was receiving an infusion of vecuronium 0.01 mg/kg/hour. An intravenous bolus dose of calcium chloride (10

Table 4. Durations of vecuronium infusion, together with total doses of vecuronium and main infusion rates at which the vecuronium was given (that is, once the rate had been adjusted to produce satisfactory control of the train-of-four) for each of the seven patients. The time to disappearance of the train-of-four after the main infusion rate had been fixed, in the four patients in which it occurred, is also given. The twitch heights (A′/A and D′/A′) on stopping the infusion and on completion of the study and the time from stopping the infusion until completion of monitoring are then noted.

Patient	Duration of infusion (hours:minutes)	Total dose of vecuronium (mg)	Main infusion rate (mg/kg/hour)	Time to disappearance of train-of-four after fixing infusion rate (hours)	Twitch heights on stopping infusion		Twitch heights on completion of study		Time from stopping infusion to completion of monitoring (hours:minutes)
					A′/A (%)	D′/A′ (%)	A′/A (%)	D′/A′ (%)	
1	6:45	16.5	0.01	0.75	0	0	90	44	12:45
2	15:20	52	0.05	2.0	0	0	77	91	10:50
3	26:20	116	0.065	2.0	0	0	54	96	21:40
4	11:44	61.5	0.065	—	8.5	0	83	96	6:42
5	25:30	42	0.015	—	10	0	50	80	21:30
6	25:30	43	0.01	—	—	—	—		— —
7	32:00	112	0.05	3.0	0	0	77	76	37:00

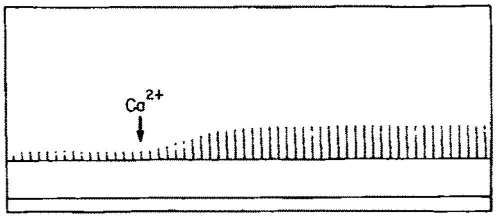

Fig. 1. The effect of calcium chloride on the train-of-four (patient 6). There is a three-fold increase in A′ and the ratio D′/A′ increased from 12 to 36%.

ml of 10% solution) was therefore given and a sudden increase in A′/A occurred (Fig. 1).

There was a wide variation in the rate of recovery of both A′/A and D′/A′ after the infusion of vecuronium had been stopped. The height of these two variables is plotted against time for each of the patients in Figs 2 and 3. It would appear that the longer the duration of the infusion (as in patients 3, 5 and 7), the longer the recovery time (Table 4).

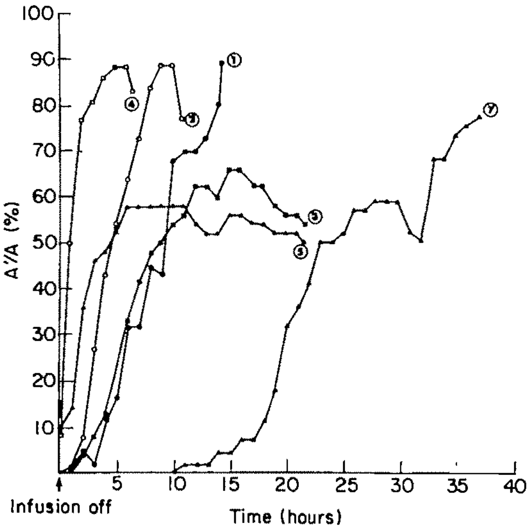

Fig. 2. The height of A′/A recorded at hourly intervals after the vecuronium infusion was stopped in each of the six patients.

The height of A′/A had not returned to 75% in patients 3 and 5 by the end of the study period but had levelled off at 54% and 50% respectively (Fig. 2), with good recovery of D′/A′ (Fig. 3). Both these patients had developed increasing peripheral oedema over this period, which affected all the limbs, including the wrists. In only one of the six patients in whom recovery was monitored did D′/A′ not reach 75%; namely, patient 1 in whom recovery of A′/A was satisfactory and in whom the twitch height had levelled off for over one hour prior to discontinuation of monitoring.

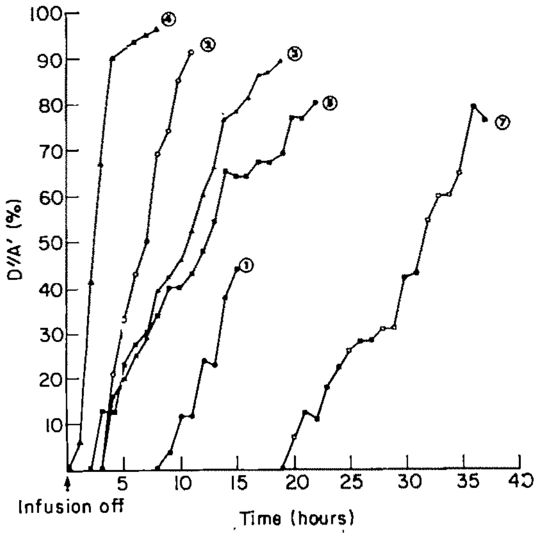

Fig. 3. The height of D′/A′ recorded at hourly intervals after the vecuronium infusion was stopped in each of the six patients.

The minimum time to satisfactory recovery, sufficient to discontinue monitoring, was 6 hours 42 minutes. This was in patient 4, who had received an infusion for only 11 hours 44 minutes; this patient had good liver function and had not received metronidazole. The maximum time to satisfactory recovery was 37 hours in patient 7, who had received an infusion for 32 hours (the longest time for which vecuronium was given). In this patient the train-of-four had disappeared 26 hours before the infusion was stopped.

No adverse cardiovascular changes were seen on administration of the bolus dose of vecuronium (0.1 mg/kg). All seven patients had periods of cardiovascular instability during the study period which were thought to be related to the effects of septicaemia: there was no evidence to suggest that the vecuronium infusion was in any way responsible. Nor was there any evidence of histamine release after administration of the bolus dose of vecuronium or at any other period of the study.

Discussion

Muscle relaxants are frequently used as an adjuvant to sedation in the ITU, particularly when the patient is severely ill and pulmonary ventilation is difficult. Conventional non-depolarising muscle relaxants, such as pancuronium and alcuronium, have always proved satisfactory when used in bolus doses in such circumstances, except when severe renal dysfunction is present. In these circumstances delayed excretion may cause long-lasting curarisation, which can be especially inconvenient when the patient is ready to be weaned from IPPV or it is important to assess cerebral status. The newer non-depolarising relaxants, atracurium and vecuronium, which do not depend primarily on the kidney for excretion from the body, have advantages in this respect, but the short duration of action of these drugs limits their use in bolus form. Atracurium infusions have already been shown to be of use in patients with renal and respiratory failure in such circumstances, since a prompt recovery from neuromuscular blockade will ensue even after several hours of infusion.[1] Concern has been expressed, however, that the main metabolite of atracurium, laudanosine, which is excreted through the kidney and is known at high doses to produce seizures in animals, may accumulate in such cases.[4] There have been no clinical reports of adverse effects in such circumstances, but a vecuronium infusion might have advantages in such severely ill patients, especially as it has minimal cardiovascular effects and histamine-releasing properties.

This study would suggest, however, that in severely ill patients a constant infusion, even of a low dose of vecuronium, may cause neuromuscular blockade from which recovery will take several hours. In addition, in contrast to work reported by Buzello and Nöldge[5] which suggested that the length of recovery is unaffected by the dose of vecuronium administered, the results we have recorded would suggest that there is indeed a direct relationship between these two variables. Such a technique would, therefore, have no advantage over the more conventional practice of bolus doses of longer-acting agents.

The prolonged recovery after an infusion of vecuronium did not seem to be related to the degree of hepatic dysfunction present, nor to the severity of the patient's condition. It is recognised that in the severely ill patient, hepatic perfusion is markedly decreased and this could delay metabolism of any drug broken down in the liver. However, in this study, it was not possible to correlate the severity of the patient's condition with the dose of relaxant required. It is interesting, nevertheless, that the two patients who were most sensitive to vecuronium were both receiving parenteral metronidazole; it has been suggested from the results of animal studies that metronidazole can potentiate the block produced by vecuronium, possibly because a breakdown product of metronidazole interferes with the pharmacokinetics of vecuronium.[6] Metronidazole alone has not been shown to have any effects at the neuromuscular junction, however, nor has it been shown to interact with pancuronium.[7]

Tobramycin is also known to potentiate neuromuscular blockade in common with all aminoglycosides, mainly by a prejunctional effect.[7] Although two of the patients who were sensitive to vecuronium received tobramycin in addition to metronidazole, another patient in the trial (case 2) also received tobramycin and did not show such sensitivity to vecuronium.

Several of the patients described in this study were either on haemodialysis or on haemofiltration whilst they were given the infusion of vecuronium, but this did not seem obviously to affect the length of time for recovery from neuromuscular blockade. For instance, patient 5 had haemodialysis whilst receiving vecuronium, but was much more sensitive to the drug than patient 3 who also received haemodialysis at this time.

It is accepted that there are limitations to monitoring of neuromuscular function over prolonged periods: for example, changes in peripheral temperature or localised oedema can affect skin impedance and hence cause difficulty in stimulation of the ulnar nerve. This probably happened in patients 3 and 5, in whom A'/A did not recover to within 70% of control over 21 hours after stopping the infusion of vecuronium (Fig. 2), although residual fade of the train-of-four was minimal (Fig. 3) and there were no clinical signs of residual neuromuscular blockade.

As vecuronium has no cardiovascular effects and minimal histamine-releasing properties, it would probably be very suitable for use in bolus doses to severely ill patients in the ITU. This study suggests, however, that it is unsuitable for infusion over several hours even in low dosage; the prolonged recovery is a marked disadvantage in this clinical situation. It would seem that a fixed-dose infusion regimen cannot be selected: full neuromuscular blockade may not be present during the first few hours of an infusion, but gradually increasing block will ensue even when the infusion is fixed at a lower dose than that suggested for surgical use (0.07 mg/kg/hour).[8] Even if the patient had continuous neuromuscular monitoring, this technique would still necessitate repeated alteration of the dose regimen if satisfactory conditions were to be achieved.

Acknowledgments

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REVIEW

Ondine's curse and the anaesthetist

S. J. MATHER

Summary

The central hypoventilation syndrome (Ondine's curse) is reviewed and discussed with emphasis on the clinical features and treatment of the disorder. It is a rare syndrome, but the anaesthetist working in a paediatric centre or in intensive care should be aware of its existence since advice and assistance in management may be required.

Key words

Ventilation; apnoea, carbon dioxide response.
Receptors; chemoreceptors.

The anaesthetist, particularly if he or she works in a paediatric centre or in intensive care, should know a little about the condition of central hypoventilation syndrome. The congenital form appears to be extremely rare, with less than 40 reported cases in the world literature, but perhaps this syndrome is more common than is generally thought. An acquired form of central hypoventilation is also seen.

This article presents an outline of the condition followed by a review of current management in which the anaesthetist is likely to be involved.

Ondine's curse

The central hypoventilation syndrome or Ondine's curse was first reported in 1955[1] and describes a group of conditions where the lungs are essentially normal but the neural control of breathing is not. The name 'Ondine's curse' derives from the story of the mythical water nymph whose husband jilted her for a mortal female and was cursed with death should he fall asleep.

Clinical features

Most cases reported in the literature have presented within a few hours of birth with cyanosis or apnoea;[2–13] this presentation has led to a diagnosis of central hypoventilation syndrome and appropriate investigation and monitoring.[2] Other modes of presentation have included convulsions and acute cardiorespiratory insufficiency.[2,12,13]

The syndrome neither seems to be commoner in one sex nor does there appear to be any association with prematurity, obstetric complication nor mode of delivery of the infant, but there may be a familial incidence.[14]

A marked disturbance of water balance with nocturnal diuresis has been noted in many of the children with central hypoventilation syndrome. In two children (P. Fleming, personal

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communication), relatively mild upper respiratory infection led to significant increases in production of antidiuretic hormone (ADH). Both these children developed convulsions associated with their infections, which were attributed to hyponatraemia.

In those cases reported in the literature no specific abnormality was found on initial examination except for hypotonia.[8,10,15] All cases were investigated subsequently (but at varying times following initial presentation) with particular reference to the possible causes of apnoea.

Association with other conditions

Other cases of respiratory difficulty and sleep apnoea were associated with ganglion-neuroblastoma, Hirschsprung's disease,[15-17] Reye's syndrome[18] and pyruvate dehydrogenase complex deficiency.[19]

Differential diagnosis

Differential diagnosis lies between congenital central hypoventilation and other conditions causing hypoventilation during sleep. These are:

- Congenital central hypoventilation
- Acquired central hypoventilation
  - encephalitis
  - bulbar poliomyelitis
  - brainstem surgery
  - cervical syringomyelia
  - sarcoidosis of the central nervous system
  - syphilis
  - dystrophia myotonica
- Obstructive sleep apnoea

Diagnostic criteria

Central hypoventilation syndrome should be suspected in any child who presents with unexplained or unexpected hypoxia, apnoea or respiratory failure. Diagnosis rests on differences between ventilation and $P\text{aco}_2$ in the awake state compared with values obtained when the patient is sleeping. Marked rises in $P\text{aco}_2$ with concomitant hypoventilation and a variable degree of hypoxia during sleep, should lead to an initial diagnosis of the central hypoventilation syndrome.

Normal respiratory control mechanisms

Neural control of breathing

This is under the control of the autonomic system but can be modified by inputs from the higher centres.

The respiratory centre

The respiratory centre is not a specific entity but rather a diffuse collection of neurones in several sites. Rhythmic respiration appears to be initiated and processed by cells in the medulla termed the *dorsal and ventral respiratory groups.*[20]

The output of the medullary centres is modified by neurones in the pons termed the *pneumotaxic* and *apneustic* centres. The pneumotaxic centre may be activated by inputs from the central or peripheral nervous systems and seems to be involved in phase switching between inspiration and expiration and, perhaps, in the integration of chemical and neural control mechanisms. Its stimulation results in an increased respiratory rate.

It seems likely that the apneustic centre may be the site of termination of inspiration, because sensory inputs which may terminate an inspiration project here. Furthermore, transection of the brainstem above the level of this centre, when combined with vagotomy, results in apneusis.

Stretch receptors

Stretch receptors are thought to be located in bronchial smooth muscle. Their input is via the vagus. In laboratory animals inflation of the lung causes a sudden reduction in respiratory rate due to inhibition of inspiration (the Hering–Breuer reflex). The rôle of this reflex in older humans is, however, controversial but it appears to be present in the neonate.

'Irritant' receptors

'Irritant' receptors probably exist between epithelial cells lining the airways and are again innervated by the vagus. They seem to be sensitive to changes in $P\text{aco}_2$, to histamine and to particulate and chemical irritants, but are also sensitive to lung distension. Their stimulation may result in cough and bronchoconstriction.

J-receptors (juxtapulmonary capillary receptors)

J-receptors are thought to be situated in the walls of pulmonary capillaries. In the cat, hyperinflation causes reflex apnoea, bradycardia and hypotension which is attributed to their stimulation. Their rôle in the control of breathing is uncertain but it is possible that they may be involved in cardiorespiratory abnormalities consequent upon an increase in lung water.

Chemical control of breathing

The three variables which are sensititively controlled are $P\text{aco}_2$, $P\text{ao}_2$ and hydrogen ion concentration. Receptors for these substances lie both peripherally and centrally, in the medulla.

The medullary chemoreceptors are very sensitive to changes in $P\text{aco}_2$ but not to changes in $P\text{ao}_2$ or pH. Carbon dioxide exerts its effect on the receptors by diffusing across the blood–brain barrier (which is impermeable to hydrogen ion) and forming carbonic acid in the cerebrospinal fluid (CSF), which has little buffering capacity. The hydrogen ion so formed ($H_2CO_3 \rightleftharpoons HCO_3^- + H^+$) is a powerful stimulus to the medullary chemoreceptors. Hypoxia potentiates this but has no direct effect on the central chemoreceptors.

Peripheral chemoreceptors

Peripheral chemoreceptors exist in the aortic arch and carotid bodies. They respond rapidly to changes in $P\text{aco}_2$, $P\text{ao}_2$ and pH. Little change in ventilation occurs as $P\text{ao}_2$ decreases until below 50 mmHg (about 6.5 kPa), when a rapid response occurs.

Ventilatory response to changes in $P\text{aco}_2$

Carbon dioxide has the most powerful effect on respiration. A 10% rise from resting $P\text{aco}_2$ can double ventilation. Most of the effect is, however, due to stimulation of the central chemoreceptors. Conversely, hypocapnia has an extremely powerful negative effect on respiration, which results in hypoventilation, even in the face of hypoxia and acidosis.

Ventilatory response to hypoxia

The ventilatory response to hypoxia is mediated only through the peripheral chemoreceptors. The dependent variable is $P\text{ao}_2$. *Mild* hypoxia stimulates ventilation little (because the fall in $P\text{aco}_2$ so produced results in overriding depression of ventilation, due to the exquisite sensitivity of the control mechanism to changes in $P\text{aco}_2$).

Sleep

Respiratory rate and minute volume are greater and show more variability in rapid eye movement (REM) sleep than in quiet sleep.[21,22] Mechanisms which overcome increased resistance to inspiration tend to maintain tidal volume but the response is much less effective during REM sleep, probably due to uncoordinated muscle action.[23] These findings may have relevance in some forms of sleep apnoea.

Pathophysiology of central hypoventilation syndrome

Several disease entities may give rise to central hypoventilation in both children and adults. Sometimes nonspecific degenerative changes are found in the medulla at postmortem and absence of the arcuate nucleus has been reported.[2] The essential pathology in these cases is failure of central chemoreceptor function or perhaps integration of chemoreceptor information,[17] which results in decreased responsiveness to carbon dioxide.

Some patients exhibit both a decrease in respiratory drive and an element of mechanical upper airway obstruction.[3] The ventilatory capacity may be normal with voluntary effort, with normal $\dot{V}/\dot{Q}$ matching. During sleep, however, the alveolar minute ventilation may be so low as to lead to hypoxia and carbon dioxide retention. This is at least partly due to $\dot{V}/\dot{Q}$ mismatch in some patients. Characteristically there is increased pulmonary vascular resistance and polycythaemia. Cor pulmonale may develop.

Central hypoventilation is seen in adults and older children as a result of discrete disease entities such as encephalitis or bulbar poliomyelitis, but the most mysterious of all is the congenital form of the disease, congenital central hypoventilation syndrome (CCHS). In the last 10–15 years it has been recognised with increasing frequency and, although still rarely reported, it has been suggested that such a syndrome may in fact be part of a more generalised disorder of autonomic function. This syndrome should be considered in any child who presents with unexplained or unexpected hypoxia, apnoea or respiratory failure.

Such a central mechanism has been postulated as a factor in the sudden infant death syndrome (SIDS),[24,25] which suggests that, at least as a contributory factor, it is far more common than is generally realised.

The central hypoventilation syndrome and SIDS

The incidence of SIDS is variously reported as 0.3–3 per 1000 live births, peaking at an age of 2–3 months. Unlike the central hypoventilation syndrome, males appear to be affected more than females.

Aetiology of SIDS

SIDS may also result from a variety of causes but cardiorespiratory apnoea is certainly a feature of some SIDS patients. Whether such apnoea is central or obstructive in origin, or a mixture of the two, remains unclear.

A depressed ventilatory response to carbon dioxide has been demonstrated in some near-miss SIDS infants, but it seems to be increased in others. Those with a markedly decreased response to carbon dioxide may be classified as the central hypoventilation syndrome but the overlap in clinical and biochemical features in some cases makes differentiation between the two syndromes difficult.

In small, premature infants, prolonged apnoeic episodes may be fatal. However, because of the developing neurophysiological mechanisms in that group, they cannot necessarily be equated with apnoea in full-term babies or infants.

SIDS patients do, however, show some similarities with the features found in some patients with Ondine's curse. Comparison is difficult, because great inter-patient variability exists in both groups and there is no discrete pathophysiological entity yet discernible to account for the various clinical observations.

Recurrent apnoea in infants

Recurrent apnoea in infants is associated mainly with prematurity but also occurs in those with hypoglycaemia, hypocalcaemia, hyperbilirubinaemia and brain injury. There may be a relationship to hypoxia, since oxygen breathing may reduce the incidence.[26] However, mean arterial P_{O_2} and alveolar–arterial oxygen difference were found to be essentially similar in apnoeic and nonapnoeic babies.[27] There may be a greater dependence on chemoreceptor drive in babies with periodic breathing, since experimentally, oxygen breathing can reduce ventilatory drive in these infants. There is also a decreased slope to the carbon dioxide response curve.

Involvement of stretch receptors is suggested by the improvement engendered by continuous positive airway pressure (CPAP) when applied to infants with recurrent apnoea. Malfunction of these receptors may be involved in the production of apnoea. Such events as are seen in premature infants may be related to sleep apnoea in older children, in particular the apparent greater dependence on chemoreceptor drive. Not all patients manifest decreased ventilation with oxygen breathing but many seem to display decreased sensitivity to rising P_{aCO_2}.

Investigation of suspected central hypoventilation syndrome (Table 1)

No published case showed any specific abnormality on routine blood, CSF and urine examination. Of more relevance were blood gas analysis and sleep studies.

Table 1. Investigation of the central hypoventilation syndrome. As many of these studies as possible should be performed with the patient awake and during sleep, over a period of at least 4 hours.

- Blood gas analysis
- Oximetry
- Transcutaneous P_{O_2}
- Capnography
- Continuous polygraphic recording of
  - EEG
  - Heart rate
  - Electromyogram
  - Electro-oculogram
  - Oesophageal manometry
  - Flow measurements and plethysmography

Specific changes found on blood gas analysis

The reported cases show a distinct difference between P_{aCO_2} when the child was awake compared with the value when he or she was sleeping. In some cases, but not in all, this was accompanied by acidosis. Hypoxia was also a frequent finding; the greatest decrease from a normal value occurred during sleep.

Rapid eye movement (REM) versus non rapid eye movement (NREM) sleep

In general, the greatest variation from normal values was seen during non rapid eye movement sleep, that is, quiet sleep. Further deterioration may occur due to excessive handling, taking of blood and after feeding. Any degree of chest infection, increase in airway resistance or decrease in compliance is likely to intensify the problem markedly.

Sleep studies

In a case of suspected central hypoventilation syndrome, observation and investigation during sleep are mandatory. Studies commonly performed should include the following: plethysmography, flow measurements, capnography, transcutaneous $P\text{O}_2$, oximetry, electroencephalogram, electromyogram, electrooculogram and heart rate. Several authors have stressed the importance of ventilatory studies during both wakefulness and sleep.[6,11,23]

Polygraphic charts (Fig. 1) will show the temporal association of any respiratory obstruction or apnoea with heart rate changes, carbon dioxide retention or hypoxia. Guilleminault *et al.*[28] showed that irregular respiration (a normal finding during REM sleep) was greater in CCHS infants than in controls. Furthermore, they also noted the occurrence of mixed central and obstructive sleep apnoea. Haddad *et al.*[15] noted a lack of heart rate variability during sleep in one infant compared to controls in whom substantial heart rate variation was seen. This would lend support to the hypothesis that CCHS is part of a greater autonomic dysfunction.

Sleep studies in published cases tend to reveal

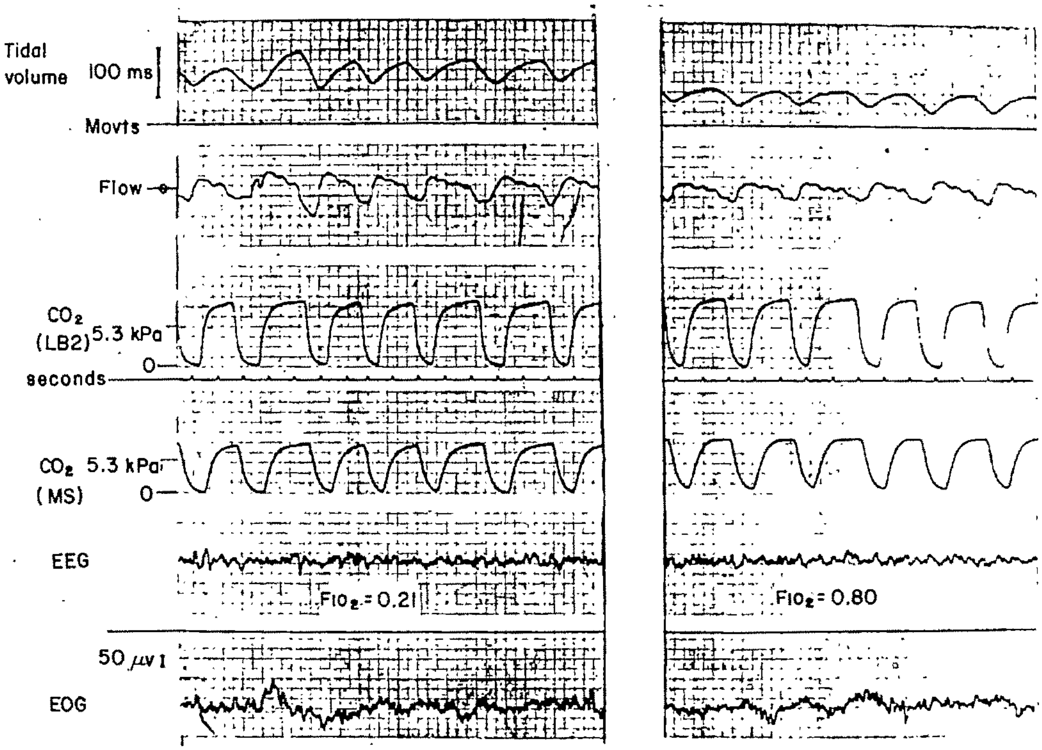

Fig. 1. Polygraphic sleep recording from a child with Ondine's curse. Note the reduction in tidal volume with oxygen breathing. LB2, Infrared gas analysis; MS, mass spectrometry; EEG, electroencephalogram; EOG, electrooculogram.

no substantial degree of hypoxia, carbon dioxide retention or acidosis during the awake state (although some have shown $Paco_2$ levels greater than the normal upper limit). In contrast, when they are asleep, CCHS infants develop hypoventilation which leads to marked hypoxaemia, followed in some cases by an improvement.

Various authors have measured hyperoxic, hypoxic and hypercapnic responses in both REM and NREM sleep. Hyperoxia is used to assess peripheral chemoreceptor function. The chemoreceptors respond to hypoxia with an increase in minute ventilation, but at normal Pao_2 there is little activity. Administration of oxygen to achieve hyperoxia would be expected to suppress ventilation, with consequent hypercapnia, if peripheral chemoreceptors are intact. This has, indeed, been found in several studies during REM and NREM sleep.[2,4,7,8,11,12]

Lack of response to increased $Paco_2$ reflects the failure of central chemoreceptors in this condition.

Indications for therapy

Most patients with the central hypoventilation syndrome are infants and small children. Therapy usually has to be instituted acutely following an hypoxic episode or cardiorespiratory collapse. Various methods of treatment have been tried.

Oxygen therapy

Low-flow oxygen therapy has been used in older children and adults with some forms of primary alveolar hypoventilation,[29–31] although carbon dioxide retention has been shown to occur when oxygen is given during sleep.[30] McNicholas *et al.*[30] used it successfully in a 58-year-old man who demonstrated no ventilatory response to hypoxia when awake, which suggested malfunction of peripheral chemoreceptors. Achievement of hyperoxia in CCHS has been shown to decrease minute ventilation during sleep. Hyperoxia may not be obtained with low-flow oxygen therapy, which is only designed to correct hypoxia. It must be stressed, however, that low-flow oxygen administration has not been used in the majority of children with CCHS.

Drug therapy

Various drugs have been tried, particularly central nervous system stimulants and drugs which act specifically upon the peripheral chemoreceptors. Naloxone has also been evaluated, with negative results.

Haddad *et al.*[15] performed controlled studies of doxapram, progesterone, aminophylline, chlorpromazine, imipramine and physostigmine in the same patient. Doxapram was the only drug of those quoted which produced any benefit. Imipramine worsened minute ventilation. Another tricyclic antidepressant, protriptyline, was used by Clark *et al.*[32] in a 13-year-old boy with central hypoventilation.

Medroxyprogesterone has recently been tried[33] with some success in CCHS but the authors reported that the drug probably has no effect when there is complete failure of ventilatory response to carbon dioxide.

Doxapram. This drug, a peripheral chemoreceptor stimulant, has been used to treat central hypoventilation in both adults and children. It is effective by intravenous infusion especially in the presence of hypoxia.[34] Enteral administration has also been tried, with disappointing results.[35] Hunt *et al.*[35] reported massive haemorrhage from the gastrointestinal tract and fatty change in the liver following prolonged intravenous administration of doxapram to children.

Almitrine. Recently, attention has been focussed on almitrine bismesylate in the central hypoventilation syndrome.[36–38] Effective doses appear to produce a significant increase in minute ventilation in both REM and NREM sleep, with concomitant improvement in blood gases during spontaneous ventilation in air. As is the case with doxapram, minute ventilation and blood gas measurements return to their pretreatment values when the drug is withdrawn. Almitrine is not yet available in the UK except under special licence.

Mechanical ventilation

The mainstay of treatment is tracheostomy with mechanical ventilation of the lungs, usually positive pressure ventilation, but negative pressure ventilators have also been used.[39] In the initial stages it will be necessary to perform all care in hospital but eventually, with training and experience, the parents may be capable of taking over all nursing care and management of the tracheostomy and ventilator. Usually only nocturnal ventilation is required; the child is attached to the ventilator upon retiring to bed.

Choice of ventilator

In hospital almost any mechanical ventilator suitable for adult or paediatric use, as applicable, will suffice. At home, however, the requirement is for a robust, simple machine which can be transported. Electrically powered rather than gas-driven machines are preferable, since a source of compressed gas is difficult to maintain without bulky cylinders or a noisy respirable air pump. We have found the Cape TC 50 to be particularly suitable for children. It is inexpensive, easy to use and requires little maintenance.

In all cases a pressure fail alarm must be provided to allow the parents some freedom from constant supervision of the child. This should also be capable of recognising high pressures, for example, to detect occlusion of the ventilator tubing. It should be *battery* operated to ensure that it will detect ventilator failure in the case of a power cut.

Condenser humidifiers are suitable for use during the daytime (when the patient is not ventilated) in the early phase following tracheostomy.

Anaesthetists are frequently called upon to advise in such circumstances and attention to such points as mentioned above will ensure that appropriate advice is given.

Further management of infants on home ventilation

The family must be provided with a self-inflating bag for resuscitation in case of ventilator failure and be properly instructed in both resuscitation, ventilation and tracheostomy care. The machine must be serviced regularly. This may entail a visit to hospital whilst the work is carried out.

Patients with central hypoventilation syndrome appear to be extremely susceptible to respiratory failure and cardiorespiratory collapse consequent upon upper respiratory or chest infections. They should always be re-admitted to the intensive care unit immediately whenever such an event occurs. Even a simple cold may lead to rapid deterioration.

The tracheostomy will need to be reviewed from time to time. The size of the tube should be increased progressively to avoid significant resistance to breathing as the child grows.

Negative pressure ventilators. Cuirass-type respirators may be suitable for patients who do not require ventilation every night.[29]

Phrenic nerve pacing (electrophrenic pacing)

Phrenic nerve pacing has been used successfully in adults and children with the central hypoventilation syndrome.[4,6,25,40–49] It is now generally felt that bilateral phrenic nerve pacing should be employed. Early disappointing results may have been due to inadequate respiratory excursion produced by pacing only one phrenic nerve. Prolonged use of such pacing can lead to nerve damage in the form of demyelination and degeneration of axons,[6,44] infection and respiratory alkalosis.[45] Table 2 indicates potential hazards of the technique.

Table 2. Early and late complications of phrenic nerve pacing.

Early failure
Surgical injury to the nerve
Infection at the electrode site or receiver site
Respiratory alkalosis
Upper airway obstruction
Late failure
Malfunction of electrode
Malfunction of receiver
Disconnexion
Diaphragmatic fatigue
Nerve damage due to electrical injury

A case is reported[44] of an infant treated with phrenic pacing who died at 8 months of age following 19 days of continuous bilateral pacing. The phrenic nerves showed axonal damage at the site of the electrode, with degeneration in the distal axons. Focal neurogenic atrophy was also observed in diaphragmatic muscle. All these lesions could be attributed to the pacing. However, several papers[25,40–43] state that good results have been obtained both in infants and adults and some authors recommend end tidal carbon dioxide measurement as a means of monitoring its effectiveness.[45]

Langou *et al.*[47] found that phrenic nerve pacing could lower the pulmonary artery pressure, which was not achieved by simple correction of hypoxaemia. They postulate that this effect is a direct result of improvement in alveolar ventilation. Reversal of cor pulmonale and polycythaemia is also described by Hyland *et al.*[48] following phrenic pacing.

Ilbawi *et al.*[40] employed percutaneous measurement of phrenic nerve conduction time and diaphragmatic action potentials to assess the feasibility of phrenic pacing in eight infants (age range 2.5–8.5 months). These authors described a technique of electrode placement through a single anterolateral thoracostomy. They placed stimulating electrodes around each phrenic nerve which was isolated with pleura and its perineural blood supply. There were no complications or deaths and the oldest survivor was 8 years old at the time of publication of their paper (October 1985). Bilateral phrenic pacing is recommended by these authors, who consider it safe and effective.

Nevertheless, there are still major problems to be overcome and one must consider that electrophrenic pacing will be required for the rest of the patient's life; the pacemaker and electrode will need to be changed at intervals.

Current recommendations imply that best results are obtained with bilateral pacing and that the phrenic nerves should be rested from time to time. Demand pacemakers, programmable through a subcutaneous receiver, are available. Since respiratory assistance is required only during sleep, it ought to be possible to tailor such a demand system to a patient's needs.

Incoordinate muscle activity may be produced by phrenic pacing. This may lead to paradoxical movement of the rib cage and even airway obstruction. Hyland *et al.*[48] emphasised this and pointed out that the normal temporal coordination of inspiratory activation of the musculature of the upper airway and diaphragm is important in preventing upper airway obstruction. In this paper the authors described a patient in whom tracheostomy was necessary to relieve upper airway obstruction engendered by the diaphragm pacing.

If diaphragm pacing is to be used it would seem prudent to monitor its effectiveness during sleep on a continuous basis. Capnography or oximetry are immediately appealing in this respect, as they are non invasive and the equipment is robust and requires the minimum of maintenance. Further, either may be used to trigger an alarm system should ventilation become inadequate and lead to hypercapnia or desaturation, respectively.

Relative value of therapies

Overall, tracheostomy and mechanical ventilation of the lungs is the most successful means of management in the long term, although a very few cases have benefited from oxygen therapy. Drug therapy has been disappointing and phrenic pacing still has a long way to go before it becomes the mainstay of treatment. However, with increasing interest in pacing technology and the development of micro-electronics and programmable pacemakers, 'state of the art' technique may soon produce a reliable system of pacing and monitoring which could be provided at moderate cost.

Prognosis

Overall prognosis in the central hypoventilation syndrome is poor both for infants with the congenital form and for older children and adults with acquired disease. All patients with Ondine's curse should be regularly followed up with polygraphic sleep studies, particularly growing children. Death is most likely to be due to respiratory infection, cor pulmonale or autonomic disturbances with cardiorespiratory failure or cardiac arrest.

The role of the anaesthetist

Anaesthetists may become involved in the emergency department when the child first presents to hospital, or later, when the diagnosis has been made.

The paediatric cases are appropriately managed wholly by paediatricians in the early stages but in many centres, help from their anaesthetic colleagues may be sought, particularly if surgery or home ventilation is contemplated. We may be asked to advise on the suitability of a particular type of ventilator, monitoring equipment or with specific problems of respiratory care. Adults who suffer from the acquired form of the disease may well be managed by anaesthetists in association with neurosurgical or general medical colleagues.

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Anaesthesia, 1987, Volume 42, pages 404–406

CASE REPORT

Delayed respiratory arrest after epidural hydromorphone

H. J. WÜST AND P. R. BROMAGE

Summary

A case is reported where hydromorphone 1 mg injected into the epidural space produced apnoea 4.5 hours later. No other narcotics had been given in the previous 10 hours.

Key words

Analgesics, narcotic; hydromorphone.
Anaesthetic techniques, regional; epidural.

Since 1980, a number of cases of delayed respiratory arrest have been reported after epidural narcotics for postoperative pain relief. Most were associated with an accidental intrathecal or subdural injection, or with combined epidural and systematic narcotic administration.[1–4] This case concerns respiratory arrest 4.5 hours after an epidural dose of 1 mg hydromorphone, where an epidural dose of 5 ml of 0.75% bupivacaine given one hour before had validated correct placement of the epidural catheter, and where no other narcotics had been given in the preceding 10 hours.

Case history

A 25-year-old, 52 kg female, height 157.5 cm, presented with episodic headache and hypertension. There was a strong family history of Type II multiple endocrine abnormality syndrome, and urinary catecholamines were elevated. A phaeochromocytoma was diagnosed and CT scan confirmed a cystic tumour in her right adrenal gland. No other abnormalities were detected. Pre-operatively she was treated with phenoxybenzamine 10 mg twice daily for 3 days, and her haematocrit was 39.2% on the day before operation. Combined epidural and light general anaesthesia were planned for right adrenalectomy, and epidural narcotics were to be used for postoperative pain relief.

Premedication consisted of 5 mg diazepam by mouth at 06:00 hours, and morphine 5 mg with promethazine 25 mg intramuscularly at 07:00 hours. At 08:00 hours a Portex epidural catheter was inserted at T_{10}–T_{11} by one of us (H.W.), using the paramedian approach. A test dose of 2% lignocaine 5 ml with 1:200 000 adrenaline was followed by two increments of 5 ml each of 0.75% bupivacaine. Sensory block extended from T_2–S_5 bilaterally. General anaesthesia was induced with thiamylal 100 mg and tracheal intubation was facilitated with 100 mg suxamethonium. Light general anaesthesia with artificial ventilation of the lungs was maintained with 0.8% isoflurane in 1:1 nitrous oxide and

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oxygen. Radial artery and pulmonary artery pressures were monitored throughout the operation and in the early postoperative period. Pancuronium 2 mg was administered before tumour removal and a sodium nitroprusside infusion was used to control hypertension for 3 minutes during manipulation of the adrenal gland, immediately prior to clamping the adrenal vessels. A top-up epidural dose of 0.75% bupivacaine 5 ml was given at 10:30 hours. The operation was completed at 11:15 hours; the patient awoke and her trachea was extubated on the operating table a few minutes later, when the blood pressure was 113/87 mmHg.

Preservative-free hydromorphone 1 mg in 10 ml saline was injected through the epidural catheter at 11:30 hours. At this time the respiratory rate was 18–20 breaths/minute, and the arterial gases with an F_{IO_2} 0.4 were P_{aO_2} 16.4 kPa, P_{aCO_2} 4.6 kPa, pH 7.35, base excess −5.2 mmol/litre. After 90 minutes the patient complained of severe itching of the face and neck, and nausea, and two doses of 0.1 mg naloxone were given intravenously. The pruritus and nausea subsided shortly afterwards.

At 12:50 hours, approximately 1.5 hours after the epidural hydromorphone, the respiratory rate was 16 breaths/minute, the blood pressure 90/50 mmHg and the arterial blood gases with an F_{IO_2} 0.4 were P_{aO_2} 12.6 kPa, P_{aCO_2} 6.1 kPa, pH 7.30, base excess −3.5 mmol/litre. The patient was drowsy and pain free.

In the intensive care unit the patient was nursed in a 30° head-up position, except when she was placed flat at hourly intervals for measurement of pulmonary artery wedge and central venous pressures. At 15:00 hours the respiratory rate was still 16 breaths/minute. One of us (H.W.) visited the patient at 15:45 hours when she responded to questions and the respiratory rate was 15 breaths/minute. Fifteen minutes later, at 16:00 hours, and 4.5 hours after the epidural hydromorphone, breathing suddenly ceased and the patient became unresponsive. There was no accompanying cardiovascular depression. Her trachea was immediately intubated by a surgical resident who happened to be watching her, and ventilation was maintained with an automatic ventilator. One hour later a trial of spontaneous ventilation was carried out. Spontaneous minute volume was 2 litres/minute at a rate of 13 breaths/minute when the patient was disconnected from the ventilator. Following intravenous injection of 0.2 mg naloxone, minute volume rose to 8 litres/minute at a rate of 16 breaths/minute, but decreased to 2 litres/minute 20 minutes later. Mechanical ventilation was therefore maintained throughout the night. Her trachea was extubated in the morning without further incident and she was discharged home in good condition on the fifth postoperative day.

Discussion

This case is instructive for four reasons. Firstly, the dose of epidural narcotic in this case was modest by most standards. Epidural hydromorphone 1 mg had been used as a standard dose for post-Caesarean section pain without ill effects in over 100 patients at the University of Colorado Health Sciences Center. However, injection in the thoracic rather than the lumbar region, lessens the distance for rostral spread to medullary and pontine receptors, and clinical reports suggest that respiratory depression is more common after thoracic epidural injection.[5,6] A smaller volume of injectate, less than 10 ml, might have been more appropriate in the thoracic region.[7,8] Also, changes of cerebrospinal fluid dynamics associated with changes of posture during measurements of pulmonary artery and central venous pressures, may have contributed to rostral spread of narcotic.

Secondly, patients with phaeochromocytoma have high circulating levels of catecholamines and these may modulate and intensify the depression caused by intraspinal narcotics.[9]

Thirdly, and with hindsight, the 33% increase of P_{aCO_2} from 4.6 to 6.1 kPa over a 1.5-hour period after the epidural hydromorphone, should have alerted us to potential trouble. (Altitude at Denver is 1603 metres and barometric pressure 84 kPa. Normal $P_{E'CO_2}$ at this altitude is 4.5 ± 0.36 kPa (our series).) However, we frequently see transient intra-operative increases of end tidal carbon dioxide in excess of this percentage change in awake patients receiving mixtures of epidural local anaesthetic and epidural narcotics, without apparent harm. In this case the resting respiratory rate was maintained at 16 breaths/minute for 4.5 hours after the hydromorphone was given, and we were lulled into a false sense of security.

Fourth, surveillance met our existing criteria for safe management of aggressive pain control.

The patient was in an intensive care unit and theoretically under close supervision. However, nursing staff were busy with adjacent patients and it was fortuitous that a surgical resident happened to be at the bedside when apnoea suddenly occurred.

The spectrum of respiratory depression from intraspinal narcotics is wide and individual cases remain disturbingly unpredictable in our present state of knowledge. This potentially lethal event contrasts alarmingly with a recently reported episode where an accidental 5-mg dose of epidural hydromorphone provided superb analgesia but quite unremarkable side effects, and where a sanguine view was taken of the relative safety of hydromorphone under such circumstances.[10] Our case demonstrates that, on the contrary, the potentially catastrophic effect of the intraspinal narcotics, including hydromorphone, must be accorded serious respect at all times.

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CASE REPORT

Unexpected difficult intubation

Asymptomatic epiglottic cysts as a cause of upper airway obstruction during anaesthesia

D. G. MASON AND K. J. WARK

Summary

Two cases of asymptomatic epiglottic cysts which presented as partial upper airway obstruction following induction of anaesthesia are described. The incidence, pathology and anaesthetic management are discussed.

Key words

Complications; intubation, tracheal.
Larynx; epiglottic cyst.

Epiglottic or vallecular cysts which present to anaesthetists, are rare, usually benign and symptomless. They are potentially dangerous[1,2] because, during anaesthesia, they may completely obstruct the larynx or the anaesthetist's view of the larynx. In the neonatal period they can cause death by asphyxiation[3–5] or continue to cause stridor and dyspnoea for months or years. Asherson[1] quoted an incidence of larger cysts of 1 in 4200 and Padfield[6] quoted a personal incidence of 4 in 5000 laryngoscopies, but the true incidence is very difficult to assess. We describe two cases of symptomless epiglottic cysts which presented after induction of anaesthesia within a period of 4 weeks to a trainee anaesthetist at The London Hospital. Fortunately, due to skilful management, this potentially dangerous complication was overcome with two different techniques.

Case histories

Case 1

A 55-year-old man was admitted for elective repair of a left inguinal hernia and banding of haemorrhoids. He was generally fit and had had a problem-free anaesthetic for an appendicectomy when aged 9 years. He was taking no regular medication and had stopped smoking 4 years before. He did not complain of difficulty in swallowing, breathing or of any other respiratory symptoms including snoring.

He was premedicated with papaveretum 15 mg and hyoscine 0.3 mg one hour pre-operatively. Anaesthesia was induced with sodium thiopentone 300 mg followed by suxamethonium 100 mg via an intravenous cannula. It was noted that ventilation of the lungs by hand was difficult via a Magill system. Laryngoscopy revealed a large 4–5-cm cyst arising from the vallecula. The cyst completely obstructed the view of the epiglottis and vocal cords and could not be lifted out of the way with the laryngoscope blade. Attempts at blind intubation were unsuccessful. The patient then commenced spontaneous respiration and anaesthesia was deepened with halothane in oxygen. An ear, nose and throat (ENT) senior registrar was called who performed direct laryngoscopy, and 20 ml of clear serous fluid were aspirated from the cyst using a

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21-gauge needle and syringe. The epiglottis and vocal cords were then exposed and tracheal intubation was performed easily with a size 9.0 cuffed oral tracheal tube.

Maintenance of anaesthesia continued uneventfully and the patient breathed spontaneously. The patient was allowed to recover on his side and the trachea extubated once full laryngeal reflexes were present. The immediate and late postoperative period passed without incident.

Case 2

A 51-year-old woman presented to dental outpatients as a day case for dental clearance. She appeared somewhat older than her 51 years and had received several general anaesthetics in the past. The last had been 7 years previously for a breast biopsy which was apparently uneventful. She smoked 15 cigarettes a day and complained of a mild, dry, morning cough. There were no other respiratory symptoms but her husband complained that she snored at night.

No premedication was administered and anaesthesia was induced with methohexitone 75 mg via an intravenous cannula. Anaesthesia was deepened with nitrous oxide, oxygen and halothane increasing to 5% while the patient breathed spontaneously. Her airway was difficult to maintain and, on laryngoscopy, a large cyst 3–4 cm in diameter was seen within the vallecula. It was soft to touch and regular in shape. Neither the epiglottis nor the vocal cords could be seen. Several attempts at intubation were unsuccessful and, because of profuse bleeding from the nasopharynx, the procedure was abandoned. The patient was allowed to wake up, after she had been turned on her side.

She was referred to the ENT department and a combined dental–ENT procedure was arranged (as an inpatient) for 2 weeks later.

Preparations were made for a difficult intubation before the case was started. The patient was premedicated with pethidine 50 mg and promethazine 25 mg intramuscularly, one hour pre-operatively. An inhalational induction with oxygen and halothane with spontaneous breathing was commenced, with full monitoring of ECG and blood pressure. Cocaine 4% was instilled into the nostrils (because of the previous problem with bleeding). Partial upper airway obstruction occurred during induction, which was not relieved with a Guedel airway. On laryngoscopy, the vallecular cyst completely obstructed the view of the larynx. A gum elastic bougie was passed blind into the trachea and a size 8.0 mm oral tracheal tube was successfully passed over it (Fig. 1).

With the aid of a Boyle–Davis gag, the surgeon was able to incise the cyst and 30 ml of a thick, cream-like material were expressed (Fig. 2). The larynx was then clearly visible and normal in appearance. The dental clearance proceeded uneventfully after nasal re-intubation of the patient. The trachea was extubated with the patient in the lateral position after laryngeal reflexes had returned. A follow-up laryngoscopy showed no recurrence of the cyst.

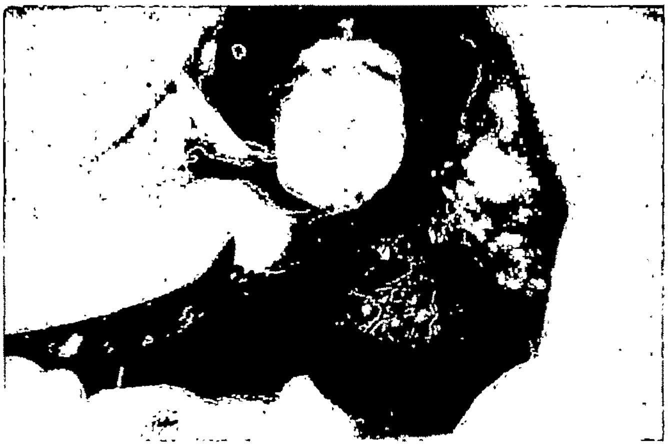

Fig. 1. Photograph of the cyst from Case 2 with Boyle–Davis gag and tracheal tube *in situ* revealing the lack of visibility of the larynx.

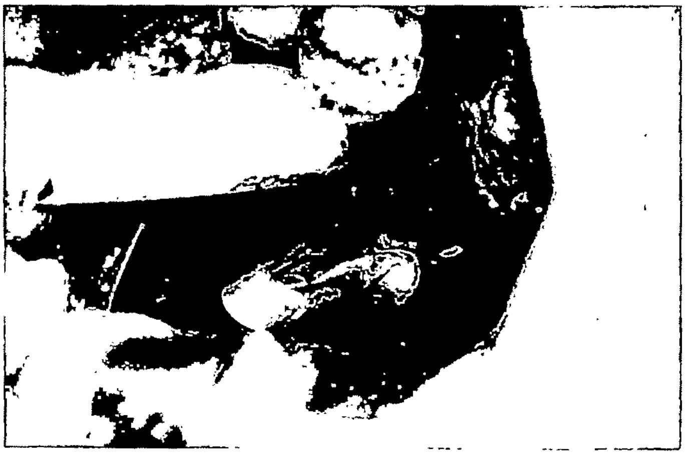

Fig. 2. Photograph of the cyst from Case 2 with Boyle–Davis gag and tracheal tube *in situ* revealing the thick, cream-like substance which was removed from the cyst.

The histological analysis of the cyst demonstrated a stratified squamous epithelial lined cyst which contained amorphous debris with patchy inflammatory infiltrate.

Discussion

The incidence of epiglottic cysts is difficult to assess as they are usually benign and asymptomatic. They may present incidentally at laryngoscopy, as in the two cases described, or be found at post mortem. If symptoms do exist, they are usually of voice change (in adults), stridor (in infants), dysphagia, pharyngeal pain or the feeling of something in the throat.[7] Rarely, epiglottic cysts may present acutely with rapidly progressive airway obstruction[1,2] due to secondary infection[8] or bleeding. This has resulted in death in both infants[4,5] and adults.[1,2] It is especially dangerous in neonates and infants since the smaller airway manifests a much greater potential for obstruction.

Cysts of the larynx are most readily classified according to their position and there are a variety of pathological processes that result in cyst formation. From a 10-year review of the Mayo Clinic, De Santo *et al.*[9] described 238 cysts of the larynx. The most common locations were the lingual surface of the epiglottis (28%) (as in the present cases), the free margin of the vocal cords (30%), arytenoid and aryepiglottic folds, the pyriform sinus and the ventricle. They further classified them into three categories based on anatomical location and probable pathogenesis: ductal, saccular and thyroid cartilage foraminal cysts.

Ductal cysts comprised 75% and were most commonly located at the true vocal cords or epiglottis. They occur because of obstruction of a submucosal gland and contain mucus which may become infected. Saccular cysts (24%) are usually congenital in origin and are found in the ventricle and lateral larynx. They originate from the epithelial surface and can contain mucus or air. They may also become infected. Other pathology which presents as laryngeal cysts may be associated with carcinoma, cautery trauma, intralaryngeal skin grafts, inflammation and fibrosis.[9]

If the diagnosis is made by indirect laryngoscopy, further investigations, prior to anaesthesia, are required to assess the extent of the obstruction. Lateral radiographs and tomograms of the neck and larynx are useful to assess the position, extent and contents of the cyst, and any laryngeal deformity. If the cyst has been diagnosed in this way, preparation can be made for a potentially difficult intubation. In this hospital, we have a difficult-intubation trolley which is readily available.

Awareness by the anaesthetist of the possibility of a difficult intubation is important in these cases, and rapid sequence induction is inadvisable. Care must be taken to protect the airway adequately prior to incision of the cyst. An awake intubation using a fibreoptic bronchoscope, or tracheostomy with adequate local anaesthesia, may be the method of choice. We prefer gaseous induction with oxygen and halothane with the difficult-intubation trolley on hand. The single most useful item is, in skilled hands, probably a gum elastic bougie. The boy scout motto of 'Be prepared' must be remembered. Also, it may be necessary to wake the patient up and start again with better preparation as in Case 2.

Acknowledgments

The authors wish to thank the ENT and Dental Departments of The London Hospital for permission to publish these cases. They would also like to thank Mrs E.A. Sparks and Miss S.A. Weber for secretarial services.

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Anaesthesia, 1987, Volume 42, pages 411–414

CASE REPORT

High frequency jet ventilation for bilateral bullectomy

G. McCARTHY, D. L. COPPEL, J. R. GIBBONS AND J. COSGROVE

Summary

This case report describes the use of high frequency jet ventilation for resection of bilateral lung bullae. Low airway pressures reduced the risk of pulmonary barotrauma. A continuous infusion of ketamine provided acceptable anaesthesia.

Key words

Ventilation; high frequency.
Anaesthetics, intravenous; ketamine.

Anaesthesia for the resection of massive lung bullae poses several problems. Intermittent positive pressure ventilation of the lungs (IPPV) may lead to a tension pneumothorax and nitrous oxide can induce barotrauma by diffusion into the bullae. It is customary practice to maintain spontaneous ventilation until the bullae can be isolated to avoid these problems, and then IPPV is instituted. In a patient with bilateral bullae such an approach must still expose one lung to the risks outlined above.

High frequency jet ventilation (HFJV), which produces only a small rise in airway pressure, is uniquely suited to overcoming these problems. It requires a total intravenous anaesthetic technique, thus fortuitously avoiding nitrous oxide. This case history describes the resection of bilateral lung bullae via median sternotomy using HFJV and an intravenous infusion of ketamine.

Case history

The patient was a 43-year-old male smoker who weighed 70 kg, with a 7–8-week history of pain in the right side of his chest and mild exertional dyspnoea. He was found to have a right-sided pneumothorax and a left-sided lung bulla. Tube drainage failed to re-expand his right lung. As a result he was moved to the Regional Thoracic Surgical Unit for pleurodesis and bullectomy. At that time pulmonary function testing indicated his forced expiratory volume in 1 second/forced vital capacity ratio to be 54%. Pre-operatively he was commenced on digoxin 0.25 mg orally daily, combined intramuscular ampicillin/flucloxacillin 500 mg 6-hourly and subcutaneous heparin 5000 units 8-hourly. His premedication consisted of intramuscular morphine 10 mg and atropine 0.6 mg one hour before surgery.

In the anaesthetic room, cannulae were inserted into a left forearm vein and the right radial artery under local anaesthetic. Following pre-oxygenation, rapid tracheal intubation was accomplished using diazepam (Diazemuls) 10 mg and ketamine 2 mg/kg intravenously and 7 mg of pancuronium. Positive inflation of the lungs

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was avoided. A size 9 mm Portex cuffed gas-monitoring tracheal tube was inserted orally and HFJV was commenced immediately via the gas-sampling side port using an Acutronic MK800 ventilator. The end of the tracheal tube was left open to the atmosphere to entrain air and was connected to an Oxydig oxygen analyser.

Initially the driving pressure was set at the minimum value of 60 kPa and at a frequency of 200 breaths/minute, with an I/E ratio of 30%. This gave a minute volume of 6 litres from the ventilator, which resulted in a $P\text{aco}_2$ of 8 kPa with a $P\text{ao}_2$ of 33 kPa.

The changes in driving pressure, minute volume and frequency and the corresponding $F\text{io}_2$, $P\text{aco}_2$ and $P\text{ao}_2$ values during the operation are shown in Table 1 and following bullectomy in Table 2. When the bullae had been resected the driving pressure was increased to 130 kPa to re-expand the lungs and allow $P\text{aco}_2$ to return to normal. Anaesthesia was maintained during the operation with a ketamine infusion at an initial rate of 4 mg/kg/hour. The ketamine infusion was reduced during the operation and finally discontinued 5 minutes before the end of the procedure. A total dose, including induction, of 524 mg was used. Labetalol was used to control the pressor effects of ketamine when the increase in systolic blood pressure exceeded 20% of baseline values.

At the end of the operation intravenous neostigmine 2.5 mg and atropine 1.2 mg were administered and HFJV was discontinued. The patient soon breathed spontaneously and opened his eyes within 2 minutes of reversal of muscle relaxation. Ventilation appeared to be adequate and the trachea was extubated. Blood gas analysis in the recovery ward was satisfactory. The patient was cooperative and his first request for analgesia came 2 hours 45 minutes after reversal, when 3 mg of morphine were sufficient. He has no recollection of his surgery nor did he suffer any emergence sequelae.

Surgical operation

Through a midsternal split, the right lung was found to be collapsed with free fluid in the pleural cavity. A large, leaking apical cyst was stapled using a TA90 instrument. The left pleura was then opened and four separate cysts were also stapled and the incision closed. HFJV provided excellent operation conditions throughout the whole procedure.

Discussion

The aim of the anaesthetic technique for resection of bilateral pulmonary bullae is to achieve the lowest airway pressure possible, consistent with adequate gas exchange. In this way the danger of pneumothorax with mediastinal shift may be avoided. To this end, Belcher[1] advised intubation under local anaesthesia and spontaneous ventilation until the chest is open. Hasenbos and Gielen[2] advocated spontaneous respiration both before and after thoracotomy in the presence of unilateral bullae. Kaplan[3] suggested independent ventilation of each lung via a double lumen tube, but again only after

Table 1. Arterial blood gases at various driving pressures prior to isolation of bullae.

Time (minutes)	Frequency (breaths/minute)	$P\text{aco}_2$ (kPa)	Minute volume (litres/minute)	Driving pressure (kPa)	$P\text{ao}_2$ (kPa)	$F\text{io}_2$
5	200	8	6	60	33	0.65
10	100	7	7	70	29	0.55
15	100	10	10	100	24	0.53

Table 2. Arterial blood gases at various driving pressures following bullectomy.

Time (minutes)	Frequency (breaths/minute)	$P\text{aco}_2$ (kPa)	Minute volume (litres/minute)	Driving pressure (kPa)	$P\text{ao}_2$ (kPa)	$F\text{io}_2$
35	100	5.8	12	120	20	0.37
55	100	6.3	11	110	39	0.55
75	100	4.9	13	130	29	0.53

the chest is open. Moreover he noted that IPPV may result in tidal volumes being delivered preferentially to compliant bullae and not to normal lung.

HFJV has been described previously by Rogers *et al.*[4] for resection of tracheal stenosis and by Normandale and Feneck[5] for coronary artery bypass grafting in bullous cystic lung disease. We are unaware of any reports of the use of HFJV in the resection of bilateral lung bullae. In this patient HFJV allowed excellent surgical access, and it is clear that although peak and mean airway pressures were not measured, the surgical conditions were such that they must have been low, so that the risk of barotrauma was negligible. To minimise this risk the driving pressure of the ventilator was kept as low as possible in keeping with acceptable levels of $Pa\text{CO}_2$ and $Pa\text{O}_2$. A small adjustment in driving pressure from 60 to 100 kPa and reduction in rate from 200 to 100 breaths/minute decreased $Pa\text{CO}_2$ from 8 to 6 kPa. The driving pressure was only increased above this once the bullae had been resected, to yield a more normal $Pa\text{CO}_2$ of 4.8 kPa by the end of the procedure. In terms of the surgical approach used, the choice lies between median sternotomy or bilateral axillary thoracotomies. Kaplan[3] stated that sternotomy is associated with a lower incidence of postoperative respiratory problems. HFJV allows sternotomy to be accomplished without undue haste and gives surgical access which cannot be matched by IPPV, since the latter tends to distend bullae and so obscure the surgical field. This can be seen in Fig. 1, in which the airway pressure was raised to distend a bulla.

Nitrous oxide is best avoided in bullectomy, since diffusion into a bulla with its subsequent expansion would risk barotrauma. Moreover, the use of nitrous oxide with HFJV creates a considerable pollution problem. HFJV using an air–oxygen mixture is satisfactory yet the question of how to provide anaesthesia remains. Inhalational agents could not be used since it is impossible to control the inspired concentration. Hasenbos and Gielen[2] selected an infusion of gamma-OH with a thoracic epidural. However, gamma-OH is not readily available in the UK. Neurolept anaesthesia is another alternative but has the potential problem of re-establishing spontaneous ventilation at the end of the procedure, together with delayed recovery. As Coppel and Johnston[6] indicated, anaesthesia with ketamine infusion is an acceptable alternative to Althesin since the latter's withdrawal. It offers good analgesia even into the postoperative period, and has already been used safely in thoracic surgery.[7] However, it has no advantage over an inhalational agent in terms of hypoxic pulmonary vasoconstriction.

After the initial induction dose an infusion was administered at a gradually decreasing rate. This resulted in a total dose of just over 7 mg/kg, consistent with previous reports of anaesthesia by ketamine infusion without nitrous oxide.

It is well known that ketamine anaesthesia can be complicated by the cardiostimulatory effect of this drug and by emergence sequelae. Lilburn *et al.*[8,9] advised use of a benzodiazepine with a long duration of action, such as lorazepam, to abolish the latter. In this case Diazemuls was used because it decreases the rigidity sometimes

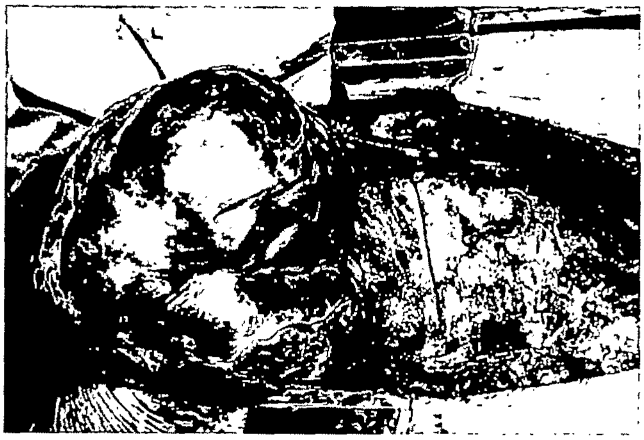

Fig. 1. Bulla distended by raised airway pressure.

associated with ketamine induction and also reduces the total dose required for anaesthesia. Diazemuls does not, however, obtund pressor responses to the combination of ketamine, intubation and surgery. Labetalol was therefore used to reduce hypertensive responses which exceeded 20% of baseline values, and proved to be effective.

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CASE REPORT

Treatment of severe accidental hypothermia using the Clinitron bed

J. M. MILES AND G. R. THOMPSON

Summary

Two cases of severe accidental hypothermia (core temperature < 28°C) rewarmed employing the Clinitron system are described. The physiological changes during hypothermia and rewarming are discussed and the current concepts of rewarming (active external versus internal) outlined. It is suggested that severely hypothermic patients can be successfully treated by rapid external rewarming using the Clinitron heated fluidised-bead bed. This method combines the advantages of rapid rewarming, minimal physiological disturbance and is non invasive.

Key words

Hypothermia.
Intensive care.

Much emphasis has recently been placed on the disadvantages of external rewarming in the treatment of severe hypothermia.[1] We report two hypothermic patients rapidly warmed using the Clinitron fluidised-bead bed,[2] which combines the advantages of a constant-temperature microclimate (range 32–40°C) and minimal skin pressure.

Case histories

Case 1

An 83-year-old woman was admitted to the intensive therapy unit (ITU) suffering from profound hypothermia (core temperature 25°C) after she was found collapsed at home. The patient was comatose and responded only to painful stimuli; her respiratory rate was 10 breaths/minute, pulse rate 55 beats/minute, arterial blood pressure 80/40 mmHg and there were signs of severe peripheral circulatory shutdown. The ECG showed junctional rhythm with wide (0.20 second) QRS complexes.

She was treated by active rewarming on the Clinitron bed (T 34°C). Over the next 14 hours her core temperature increased to 36.4°C at a rate of 1°C (SD 0.2) per hour. In order to compensate for the peripheral vasodilatation and fluid shifts into the extravascular compartment associated with rewarming, 4.8 litres of dextrose/saline with added potassium (150 mmol total) were infused during the first 24 hours. Dopamine 5 μg/kg/minute was employed to maintain cardiac output and increase renal perfusion. Her cardiovascular status remained stable apart from a short episode (30 seconds) of bradycardia, at a core temperature of 33.5°C, which spontaneously reverted to sinus rhythm with left bundle branch block. The patient

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breathed spontaneously throughout rewarming. Supplementary oxygen (40–60%) was required initially via a facemask and this was gradually reduced to air by day 3.

The patient regained consciousness within 24 hours, was fully alert by day 4 and was discharged home 5 weeks later without any permanent disability.

Case 2

A 71-year-old woman was admitted to the ITU after she was found unconscious at home. The patient was profoundly hypothermic (core temperature 25.6°C) and comatose. Her respiratory rate was 13 breaths/minute, pulse rate 36 beats/minute, arterial blood pressure 150/80 mmHg and clinically the patient had signs of peripheral circulatory inadequacy. She was treated by rewarming on the Clinitron bed (initial T 36°C, increased to 40°C after 3 hours).

Immediately after transfer to the ITU she had an asystolic episode which responded promptly to external cardiac massage, intravenous atropine 0.3 mg and oxygen by facemask. She then remained in a sinus bradycardia with frequent atrial and infrequent ventricular ectopics, but maintained an adequate cardiac output.

Over the following 12 hours her core temperature rose to 36.4°C at a rate of 0.9°C (SD 0.2) per hour. Dextrose/saline 4.5 litres with added potassium (130 mmol total), plus dopamine 5 μg/kg/minute, were infused over the first 24 hours. The patient's blood pressure remained stable and her urine output was normal. The pulse rate started to increase at a core temperature of 31°C and within 4 hours stabilised at 80 beats/minute (SD 25). Several episodes of hypoglycaemia, during the first 48 hours, were treated with boluses of dextrose 50% (total 80 g). She maintained adequate spontaneous ventilation; oxygen (40–60%) was added initially and gradually reduced to air by the end of day 2.

Table 1. Details of haemodynamic, biochemical and haematological values.

		Time (hours)								
	Patient	0	2	4	6	8	10	12	14	16
Core temperature (rectal) (°C)	1	25	26.6	29.2	32.1	34.3	35.3	35.8	36.2	36.5
	2	25.6	27.8	29	31.2	33.3	35.6	36.4	37.3	37.2
Mean arterial pressure (mmHg)	1	56	60	76	73	60	73	82	65	66
	2	100	90	68	62	70	67	72	63	65
Pulse rate	1	55	52	90	80	110	80	90	100	105
	2	36	50	53	71	83	98	98	105	100
Mean urine output (ml/hour)	1				100					
	2				95					
Respiratory rate	1	10	12	22	20	20	16	20	18	18
	2	13	14	16	18	16	22	22	24	24
Po_2 (kPa) (Fio_2)	1	18.5	11.0	4.5	9.1	24.0	10.0	9.2	10.7	9.4
		(0.4)	(0.4)	(0.4)	(0.6)	(0.6)	(0.4)	(0.4)	(0.4)	(0.4)
	2	5.2	10.3	10.0	9.0	11.9	14.1	12.0	11.0	12.9
		(0.4)	(0.6)	(0.4)	(0.4)	(0.4)	(0.4)	(0.35)	(0.35)	(0.35)
Pco_2 (kPa)	1	3.3	3.7	3.6	4.1	4.5	4.9	4.2	4.6	4.7
	2	3.6	3.8	3.8	4.2	4.0	4.2	4.0	4.0	3.9
pH	1	7.46	7.41	7.41	7.43	7.39	7.38	7.41	7.40	7.40
	2	7.37	7.37	7.36	7.33	7.35	7.33	7.39	7.38	7.38
HCO_3/base deficit (mmol/litre)	1	21.3/4.7							22.3/1.8	
	2	18.3/8.6							17.5/6.7	
Serum sodium (mmol/litre)	1	131		129						
	2	135			135		136			
Serum potassium (mmol/litre)	1	3.3	2.7	3.4	4.3	4.4	5.1	5.0	5.2	4.9
	2	3.4	4.0	3.9	4.4	4.3	4.0	5.0	4.9	5.3
Serum glucose (mmol/litre)	1	7.0	6.0	8.5		5.5		6.0	7.0	7.0
	2	6.0	6.5	5.0	3.5	3.0	4.0	2.5	3.0	4.5
Hb(g/litre)/ haematocrit	1	122/0.36							107/0.31	
	2	85/0.26							84/0.25	

Arterial blood gases were measured using the ABL4. All Po_2, Pco_2 and pH values were corrected for temperature. Core temperatures were monitored by a thermocouple rectal probe (Roche). Central venous and arterial lines, a urinary catheter and a nasogastric tube were inserted in both patients.

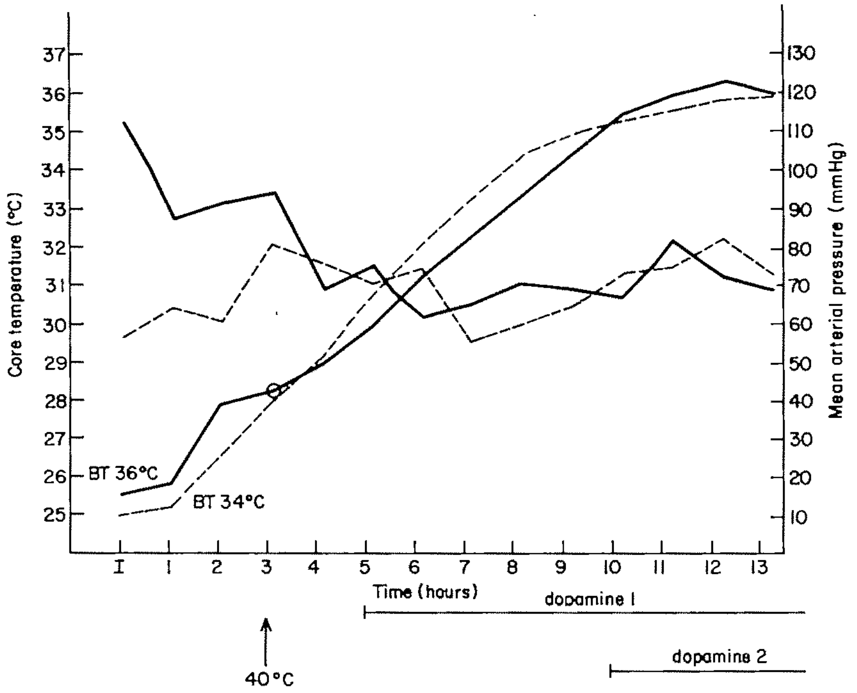

Fig. 1. Plots of body core temperature and mean arterial pressure demonstrating smooth gradual rewarming and cardiovascular stability. - - -, Patient 1; ——, patient 2. BT, Bed temperature.

After 20 hours the patient regained consciousness. Her mental state improved over the following 3 days and she was discharged home 4 weeks later with no neurological deficit.

Haemodynamic, biochemical and haematological data for both patients are presented in Table 1 and plots of core temperature and mean arterial pressure in Fig. 1.

Discussion

Accidental hypothermia (body core temperature < 35°C) is known to be associated with a high mortality (40–80%).[3] The most important prognostic factors appear to be pre-existing pathology,[4] age and the core temperature on admission.[5]

Various active core rewarming methods—peritoneal irrigation, gastro-intestinal balloons, inhalation of warm humidified gases, extracorporeal blood rewarming—have been recommended for minimising haemodynamic and metabolic disturbances associated with rewarming.[5,6] However, they are invasive, predispose to iatrogenic complications and require specialised equipment and skills. We have used the Clinitron bed for rapid external rewarming at a steady rate of 1°C (SD 0.2) per hour. Both patients made a complete recovery despite unfavourable prognostic factors of advanced age and profound hypothermia (T < 28°C).

Active external rewarming has been reported to cause an afterdrop in core temperature, which results in intramyocardial temperature gradients and increased myocardial irritability together with exacerbation of the systemic acidosis.[1] We observed no afterdrop in rectal temperatures; furthermore, the incidence of dysrhythmias was minimal. Both patients developed a degree of hypotension secondary to peripheral vasodilatation and relative intravascular volume depletion. By carefully controlled intravascular expansion we were able to maintain cardiovascular stability. The resulting reduction of afterload and haemodilution, by counteracting the increased blood viscosity at low temperatures, puts less strain on the myocardium. We used low-dose dopamine to maintain adequate renal perfusion and urine output.

The respiratory rate of both patients rose as the patients rewarmed. The arterial carbon dioxide tension and pH remained remarkably stable. The transient hypoxaemia during the early stages of rewarming (presumably due to an increased oxygen consumption and to intrapulmonary shunting), was controlled by added inspired oxygen. It would therefore appear that the rate of rewarming used in these patients resulted in minimal homeostatic disturbance.

The plasma glucose concentration tends to be labile in hypothermic patients, although hyperglycaemia secondary to insulin resistance and raised catecholamines of the stress response, is more usual. Our second patient suffered several hypoglycaemic attacks, which were promptly treated with hypertonic dextrose. Both patients had increased plasma cortisol levels.

Because of the high incidence of gastric ulceration and pneumonia associated with hypothermia,[3] prophylactic ranitidine and benzylpenicillin were given in both cases. Finally, despite the very poor initial appearance of their skin, neither patient developed pressure sores.

We suggest that the Clinitron system is a method of rapid external rewarming which combines ease of access (for monitoring and resuscitation), pressure area safety and patient acceptability with minimal physiological disturbance during rewarming and thus compares favourably with other methods currently employed.[7–9]

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APPARATUS

A new ventilator for closed breathing systems

A. TWIGLEY, M. K. CHAKRABARTI AND J. G. WHITWAM

Summary

A new electronic ventilator for application during closed breathing system anaesthesia is described (OAV7700, Ohmeda). It is easy to use and can be mounted on any conventional anaesthetic machine. It has several desirable features, such as a simple control and display panel, appropriate alarms and a patient breathing system that can be autoclaved. Observations were made on seven patients undergoing major surgery. Anaesthetic gases were introduced at 1, 2 and 4 litres/minute at three points in the breathing system, one conventionally in the closed system, one at the ventilator and one in the tracheal tube. Carbon dioxide homeostasis was maintained during this procedure. The application of fresh gas flows of 2 and 4 litres/minute in the tracheal tube allowed a small but significant reduction in the mean tidal volume. However, it was concluded that a fresh gas flow of 1 litre/minute applied conventionally either into the closed system or at the ventilator, provided optimal conditions.

Key words

Equipment; ventilators.

The development of modern ventilators has depended largely on the application of electronics. These ventilators are complex and expensive but the breathing system is conventional, with separation of inspiratory and expiratory gases, and they are not readily adaptable for use in circle systems. However, closed breathing system anaesthesia is practised widely throughout the world. Hitherto, ventilators used with circle systems tended to belong to a previous generation designed before the development of electronic control and the requirement for warning devices to be incorporated.

The OAV7700 ventilator (Fig. 1) (Ohmeda; British Oxygen Company) is an electronically controlled, pneumatically driven ventilator designed for use during circle breathing system anaesthesia. It uses a microprocessor for control, display and alarms.

General description

The pneumatic drive unit (Fig. 2) operates the patient bellows in response to the settings of the tidal volume (V_T), frequency (f), inspiratory to expiratory ratio (I:E) and inspiratory pause controls. The V_T control adjusts a potentiometer which provides a signal for a comparator circuit in the electronic control unit. When the bellows expands to accommodate the selected V_T, the comparator circuit de-energises the up

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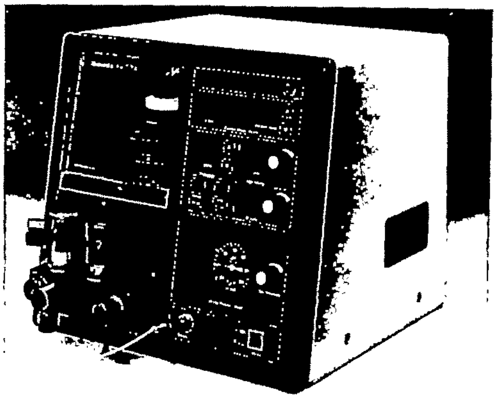

Fig. 1. New closed circuit ventilator (OAV7700, Ohmeda).

solenoid S1 to stop the gas flow to the bellows drive unit. The down solenoid S2 is energised and the gas flow is allowed to push the bellows down by means of the drive unit to deliver the required V_T in the inspiratory phase. The f control adjusts the inspiratory time, i.e. the period between the end of an expiratory phase and the end of the next inspiratory phase. In other words, it functions by controlling the time for the compression of the bellows which drives the respiratory gas into the patient's lungs.

The I:E ratio control functions as a flow control device by which the gas flow in the drive unit, and hence the rate of compression of the bellows, is adjusted. After the delivery of V_T

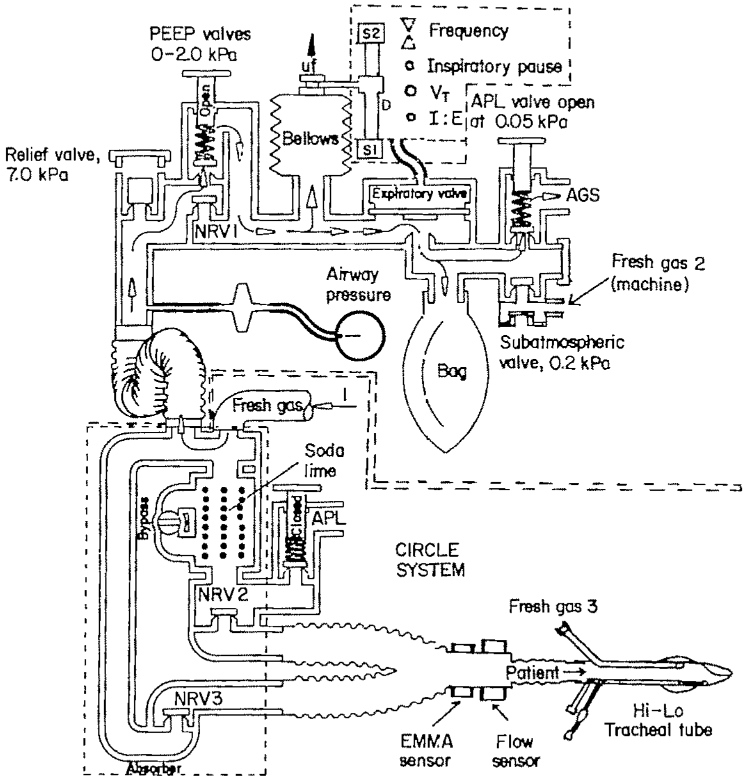

Fig. 2. Ventilator circuit for OAV7700 (Ohmeda). S1, Up solenoid; S2, down solenoid. The expiratory valve is pneumatic, and controlled with a solenoid. Sites of application of anaesthetic gases into the circle system are indicated as follows: fresh gas 1, conventional site of application; fresh gas 2 and 3, potential sites not recommended by the manufacturers. NRV, Non-return valves; APL, adjustable pressure-limiting valve; AGS, access to gas scavenging.

during the inspiratory phase, when the bellows bottoms completely, it is allowed to expand during the early part of expiratory phase at a preset time of approximately 1 second for the maximum excursion, i.e. 1.5 litres. The inspiratory pause control sends a signal to the control unit which incorporates a microprocessor; it adds an extra 25% to the inspiratory time and holds the bellows in the compressed condition for this additional time before the expiratory phase is initiated. A solenoid valve controls the flow of gas to the pneumatically operated expiratory valve. All the solenoid valves are controlled by a microprocessor control unit.

The V_T can be varied between 100 and 1500 ml, the I:E ratio from 1.0:0.1 to 1:99, f from 3 to 60 breaths/minute and positive end expiratory pressure (PEEP) from 0 to 2 kPa.

Inspiratory phase

The length of the bellows as it expands, is determined by the setting of the V_T control. When the bellows is expanded to the selected V_T position, the voltage across the potentiometer equals the reference voltage input to the preset V_T comparator. The microprocessor on the central pressure unit (CPU) board recognises from this, that the bellows has reached the set V_T position and energises the bellows brake. After a delay the brake is de-energised and the bellows begins to be compressed at a rate that depends on the I:E ratio control setting. At the same time the expiratory valve is closed. The gas from the bellows forces the non-return valve (NRV1) to open and it flows through the non-absorbing limb of the circle system and passes to the patient via another non-return valve (NRV3). If the patient's airway pressure exceeds the set maximum airway pressure then the audible alarm produces an alternating tone and the excess pressure legend is shown. When this condition occurs the inspiratory phase is terminated, any pause setting is cancelled, and an expiratory phase commences. The patient is also protected from overpressure by a pressure relief valve set to 7 kPa. When the inspiratory pause is selected via a switch on the front panel, the bellows is held in the fully compressed position for 25% of the inspiratory time. This affects the I:E ratio, as the microprocessor tries to maintain the preset frequency by adjusting the expiratory time.

Expiratory phase

At the end of the inspiratory phase, the microprocessor initiates the expiratory phase to de-energise the down solenoid valve S2 and the expiratory valve and to energise the up solenoid valve S1 after a delay. As the bellows expands the patient's expired gas flows from the patient via a flow transducer (FT) and a non-return valve (NRV2) to the soda lime absorber which removes the carbon dioxide. As the patient exhales, the gas flow is measured by a flow transducer and the signal derived from it passes to the microprocessor where the V_T and minute volumes are calculated and then displayed on the front panel. If the minute volume falls below the low minute volume alarm level, the alarm is set off. However, in the absence of the flow transducer, the display will be blanked out but the ventilator continues to function normally.

The gas from the absorber is mixed with the fresh gas which is supplied into the circle system. The gas then flows to the adjustable PEEP valve which opens to allow the gas to flow to the reservoir bag and to the bellows. The pressure in the system is monitored by the airway pressure gauge with high and low pressure sensors which are adjustable.

When the bellows rises at a rate which cannot be matched by the combined expired and fresh gas flow, the reservoir bag will be emptied and if the pressure falls below −0.2 kPa, air enters the bellows through a subatmospheric valve.

Patient synchronisation

The flow sensor is linked through the microprocessor to the down solenoid S2 so that an inspiratory phase cannot be initiated while gas is moving out of the patient. Thus, if spontaneous breathing occurs, machine inflation is prevented during spontaneous expiration. Hence the patient does not breathe out of phase with the ventilator.

Open system mode

This ventilator is designed principally for ventilation during closed system anaesthesia, but it can also be used as a conventional open system ventilator. In the open system mode the fresh gas supply is connected via an adaptor at the subatmospheric valve. The patient hose is connected to a non-rebreathing valve with an outlet to the atmosphere.

Manual mode

In the manual mode, the reservoir bag is compressed manually to force gas into the patient whilst the bellows is held in the closed position. Two adjustable pressure-limiting valves (APL) with a minimum opening pressure of 0.05 kPa are incorporated, one in the ventilator and one in the circle system. The fresh gas supply is increased to compensate for the gas lost through the APL valve. In the manual mode only the V_T and minute volumes are monitored.

Clinical application

The ventilator was used in the closed breathing system mode and was mounted on a conventional anaesthetic machine for the supply of anaesthetic gases.

Patients and methods

Observations were made on seven adult patients undergoing major hepatobiliary surgery. Their ages and weights were in the ranges 58–72 years and 55–95 kg, respectively. All were in ASA grades 2 or 3. The patients received lorazepam 2–5 mg orally for overnight sedation and papaveretum 15–20 mg, hyoscine 0.3–0.4 mg and droperidol 5 mg intramuscularly 90 minutes before the induction of anaesthesia.

A peripheral vein and a radial artery were cannulated using topical analgesia and a standard lead II electrocardiogram was recorded. Anaesthesia was induced with intravenous thiopentone 4 mg/kg and diamorphine 0.1 mg/kg while the patients' lungs were ventilated with oxygen and paralysed with pancuronium 0.1 mg/kg. Subsequently during surgery which lasted from 3–6 hours, diamorphine and pancuronium were administered in incremental doses to maximum amounts of 0.3 mg/kg and 0.15 mg/kg, respectively. Intubation was achieved with Hi-Lo tubes (Mallinckrodt 86615; 8–9.0 mm size). Two cannulae were then introduced into the right internal jugular vein and a further one into a second peripheral vein.

The arterial, central venous and airway pressures (measured near the carina from one of the catheters in the tracheal tube) and the electrocardiogram and heart rate were observed and recorded continuously (Roche monitoring system).

The concentration of isoflurane was measured by placing a sensor (Emma, Engström) near the mouth between the Y-piece of the ventilator and the tracheal tube (Fig. 2). The system was precalibrated with a refractometer (Riken). The flow sensor for the measurement of tidal and minute volumes was placed in a similar position (Fig. 2). Arterial blood gas tensions were measured with a radiometer system (ABLII).

Experimental protocol

The initial setting of the ventilator was at $f =$ 12 breaths/minute with an I:E ratio of 1:2. A fresh gas flow of 4 litres/minute of approximately 33% oxygen in nitrous oxide and 1–2% isoflurane, depending on the cardiovascular response of the patient, was applied conventionally into the circle system (fresh gas 1, Fig. 2) and the tidal volume set to provide a $Pa\text{CO}_2$ between 4.0 and 5.0 kPa. Thirty minutes were allowed to elapse to reach equilibrium and the tidal volume was then adjusted to provide a near normal $Pa\text{CO}_2$ (Table 1).

After this, three flows of anaesthetic gas at 1, 2 and 4 litres/minute were applied into the circle in the conventional and recommended way (Fig. 2), at the machine before the reservoir bag, and also via one of the catheters in the Hi-Lo tube (not recommended by the manufacturer). The flows and the site of application of anaesthetic gases were selected randomly.

The f of 12 breaths/minute and the I:E ratio of 1:2 were maintained constant throughout, while V_T was adjusted to give an approximate constant end tidal carbon dioxide concentration and $Pa\text{CO}_2$. At least 10 minutes were allowed after each change, when all the relevant measurements and blood samples were taken.

In order to measure V_T, end tidal carbon dioxide, oxygen and isoflurane concentrations when the supply of anaesthetic fresh gas was inside the tracheal tube, it was necessary to discontinue the fresh gas supply for at least half a minute in order to avoid the continuous flow of anaesthetic gases through the sensors during the expiratory phase. The volume of gas in the reservoir bag was sufficient to prevent air entrainment during this period.

Table 1. Data from seven patients, expressed as mean (SD). The frequency of ventilation was 12 breaths/minute and the oxygen and isoflurane concentrations measured near the mouth were constant.

Anaesthetic fresh gas											
Site of application	Flow rate, litres/minute	Tidal volume, ml	Airways pressure, kPa	End tidal CO_2, %	Mean arterial pressure, mmHg	Heart rate, beats/minute	Patient's temperature, °C	$Pa\text{O}_2$, kPa	$Pa\text{CO}_2$, kPa	Arterial pH	Base excess, mmol/litre
Circle system	4	503 (45.3)	1.50 (0.03)	4.06 (0.03)	74 (11.8)	76 (13)	36.5 (0.4)	18.00 (2.06)	4.30 (0.30)	7.430 (0.060)	0.28 (1.52)
	2	493 (52.8)	1.46 (0.03)	4.15 (0.43)	76 (14.6)	78 (9)	36.4 (0.6)	18.90 (1.83)	4.44 (0.23)	7.450 (0.031)	0.34 (1.69)
	1	485 (47.8)	1.46 (0.03)	4.13 (0.53)	76 (11.7)	79 (10)	36.8 (0.3)	19.39 (1.90)	4.56 (0.40)	7.441 (0.040)	0.45 (1.66)
Machine	4	465 (55.8)	1.51 (0.02)	4.30 (0.42)	75 (11.3)	79 (9)	36.6 (0.03)	16.64 (2.70)	4.50 (0.34)	4.34 (0.061)	−0.13 (1.34)
	2	485 (45.9)	1.48 (0.03)	4.28 (0.38)	76 (15.2)	77 (8)	36.6 (0.3)	18.24 (2.07)	4.46 (0.36)	7.460 (0.042)	0.13 (1.32)
	1	481 (37.6)	1.48 (0.02)	4.20 (0.49)	75 (10.6)	78 (9)	36.5 (0.7)	19.28 (2.60)	4.53 (0.37)	7.461 (0.040)	0.88 (1.72)
Tracheal tube	4	417* (58.2)	1.36 (0.04)	4.11 (0.38)	73 (15.6)	82 (8)	36.2 (0.4)	17.47 (3.26)	4.30 (0.45)	7.462 (0.050)	0.79 (1.49)
	2	434* (57.4)	1.40 (0.03)	4.23 (0.38)	75 (19.6)	79 (7)	36.3 (0.5)	19.62 (3.06)	4.40 (0.36)	7.470 (0.051)	1.14 (2.17)
	1	468 (52.1)	1.42 (0.03)	4.16 (0.53)	74 (8.7)	79 (7)	36.3 (0.7)	20.16 (3.66)	4.32 (0.52)	7.470 (0.062)	0.93 (1.97)

* $p < 0.05$.

Statistical analysis was performed using two-way analysis of variance followed by paired *t*-tests where appropriate, and a p value of less than 0.05 was accepted as significant.

Results

The results are summarised in Table 1. Figure 3 illustrates the flow waveform obtained with the ventilator set at $f = 10$ breaths/minute.

Effect of site of application and flow rates of anaesthetic gases

There were no statistically significant differences among any of the observations until the fresh gas was supplied into the distal end of the tracheal tube, when it was found that with fresh gas flows of 2 and 4 litres/minute the V_T required to maintain a normal $Pa\text{CO}_2$ was significantly less than at 1 litre/minute. With a fresh gas flow of 1 litre/minute the V_T requirement did not differ significantly among any of the sites of application, or from the requirement with the higher flows supplied at the machine and into the circle system.

Interaction of controls

This was not studied in detail, but the following general observations were made. Starting with an I:E ratio of 1:2 and an f of 12 breaths/minute, when V_T was increased the inspiratory time lengthened and the I:E ratio increased and *vice versa*. Alternatively, at a constant V_T and with an initial I:E ratio of 1:2, an increase in the ventilation frequency, by shortening the cycle time, also caused a relative increase in the inspiratory time and hence again an increase in the I:E ratio, and *vice versa*. However, once the V_T and f values have been changed, the I:E ratio can be reset independently to the starting value.

Conclusions

This new ventilator contains a microprocessor which allows it to meet the requirements of the system. Thus it provides *controls* for V_T, f, I:E ratio and PEEP; *displays* of f, V_T, minute volume, I:E ratio and airway pressure; and *alarms* for low and high airway pressures, loss of minute volume, driving gas pressure and electrical failure. It automatically synchronises

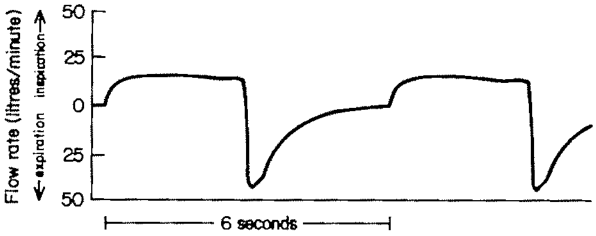

Fig. 3. Flow waveform of the ventilator with respiratory rate set at $f = 10$ breaths/minute, with $V_T = 0.6$ litres. The inspiratory flow is seen to be constant during compression of the bellows.

with the patient's respiration if spontaneous breathing occurs. The complete breathing system including the bellows is easily removed for autoclaving.

The interactions between the V_T and f controls with the I:E ratio setting are not a problem. However, since the latter is displayed it allows perception of this interaction and this could be disconcerting to users unfamiliar with this phenomenon. Without the flow sensor, tidal and minute volumes are no longer displayed. Unfamiliar users had difficulty in assessing a correct tidal volume, but this is a problem with many such systems and merely requires adequate experience with a particular system.

That the higher gas flows applied in the tracheal tube allowed the use of a smaller tidal volume, is presumably due to more efficient scavenging of the apparatus deadspace. However, in practice this was considered to complicate the system and there is little advantage over the lower gas flows (1 litre/minute) applied into the circle system in the conventional way.

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Professor John Clutton-Brock, MA, MB, BChir, FFARCS, DA
1912–1986

Professor John Clutton-Brock, the first Professor of Anaesthesia in the University of Bristol, died peacefully on 13 October 1986, aged 73.

He was born in Surrey in 1912, the son of an eminent barrister, Arthur Clutton-Brock, and Evelyn (*née* Vernon Harcourt). He was educated at Charterhouse School, Trinity College, Cambridge and St Bartholomew's Hospital.

After graduation he undertook his first post as a doctor in Bridgwater, Somerset and, after a period in the Royal Army Medical Corps, went into general practice in Lincoln. Anaesthesia became a consuming interest and he devoted more and more of his time to this specialty as a general practitioner anaesthetist. His obvious talents were acknowledged and he was appointed Consultant Anaesthetist to Lincoln County Hospital. Later he moved to Selly Oak Hospital in Birmingham, but the academic urge brought him back to the West Country when he was appointed Lecturer in Anaesthesia to the University of Bristol in 1953. He rose after a few years to the rank of Senior Lecturer and Head of Department, and it was quite natural that he should be appointed to the first Chair in Anaesthesia when it was created in Bristol in 1965. He held this post with distinction and was appointed Professor Emeritus upon his retirement in 1975.

John Clutton-Brock was renowned as an innovator and he possessed that profoundly inquisitive nature which is the hallmark of the true researcher. His particular interest in the functions of the brain led him to become one of the early clinical experts in electroencephalography and he applied this knowledge to determine brain function during artificial perfusion in the very early days of open heart surgery. He devoted much of his research to safety in anaesthesia and finding that delicate balance between light anaesthesia and the avoidance of awareness. He investigated the barbiturates, which are still very commonly used to induce anaesthesia; as a result, doses were reassessed and he ensured that more attention was paid to reducing the neurological effects of surgical stimuli and pain.

John Clutton-Brock will be sadly missed by all who knew him, as he was a gentle, humorous, widely educated man whose company was always stimulating, for he could converse knowledgeably on virtually any subject, particularly the arts, music and pottery.

He is survived by his wife Joy and by five children, one of whom, Tom, is following in his father's footsteps in anaesthesia.

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P.J.F. BASKETT

Dr Geoffrey Kaye, MD (Melb), MBBS, FFARACS, FFARCS, DA (R.C.P. & S.)
1903–1986

The death is announced of Dr Geoffrey Kaye, a pioneer specialist anaesthetist in Australia, and first Honorary Secretary of the Australian Society of Anaesthetists 1934–46. Dr Kaye was virtually the founder of this Society, for it was he who, by suggestion, consultation and correspondence, arranged the foundation meeting in Hobart, Tasmania, in January 1934. He lived to deliver the opening address at the meeting in Sydney which celebrated the Society's 50th year in January 1984.

Dr Kaye's enormous contribution to the development of organised anaesthesia in Australia was recognised by election to Life Membership of the Society in 1964, and by election to Honorary Fellowship of the Faculty in 1978. In 1974 he received the prestigious Orton Award, the Australasian Faculty's highest honour.

Dr Kaye was born in Melbourne in 1903, but received his education in England, first at Peterborough Lodge Preparatory School, and then at Gresham's School, Holt, Norfolk. He returned to Australia and graduated from the University of Melbourne Medical School in 1926. Having decided to specialise in anaesthetics he carried out research work first at the Baker Institute of Medical Research at the Alfred Hospital, Melbourne, and then for a year, in 1929–30, at the physiology department of St Thomas's Hospital, London. He met Dr Zebulon Mennell at St Thomas's and this meeting led to Dr Mennell's presence at the first Annual General Meeting of the Australian Society in Melbourne in 1935. Dr Mennell was the Society's first invited overseas guest lecturer. At this meeting Dr Mennell, as Treasurer of the Association of Anaesthetists of Great Britain and Ireland and representing the Association, proposed affiliation of the Australian Society with the Association. This affiliation, strongly supported by Dr Kaye, virtually led to the development of the specialty in Australia along British paths, rather than those of America, although it was from Dr F.H. McMechan of the United States that Dr Kaye received the impetus towards foundation of the Australian Society.

Dr Kaye's pioneering work in Australia was recognised in Britain with the award of a foundation Diploma in Anaesthetics (R.C.P. & S.), and by election to Fellowship of the Faculty of Anaesthetists of the Royal College of Surgeons of England, in 1949.

Geoffrey Kaye was known to most of the older generation of English anaesthetists through his numerous early visits to England, his prolific contributions to journals and his vigorous correspondence. His name is frequently to be found in publications on anaesthetic apparatus, for this subject was his most intense and lasting interest. It was this interest which led to foundation of the now famous museum of the Australasian Faculty which bears his name. It is pleasant to record that, in the year preceding Dr Kaye's death, much cataloguing and restorative work has been carried out in the Geoffrey Kaye Museum by the present Dean of the Australasian Faculty, Dr R.A. Smallwood, Dr T.C.K. Brown of the Royal Children's Hospital, Melbourne, and Dr R. Westthorpe, who is the newly appointed Curator of the museum.

Dr Kaye died on 28 October 1986.

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G. WILSON

Nitrous oxide and pregnancy

The paper by Crawford and Lewis (*Anaesthesia* 1986; **41:** 900–5) adds to the growing body of evidence that there appears to be no association between the use of nitrous oxide as an anaesthetic during pregnancy, and fetal malformations. This accords with our own prospective and retrospective studies[1] and a recent case report.[2] None of these studies can prove a negative, but their consistency gives growing confidence that this hazard does not seem, in practice, to be appreciable.

We would like, however, to set the record straight on two points. Firstly, it is untrue to say that there is 'no measurable effect on DNA synthesis, at least in bone marrow, unless exposure continues for 6–12 hours'; (Editorial, *Anaesthesia* 1986; **41:** 897–9). Abnormal dU suppression tests have, in fact, been reported following anaesthetics that lasted less than 2 hours.[3,4] Secondly, it should be stressed that nitrous oxide is a proven, albeit weak, teratogen in rats,[5–7] a point which escapes mention both in the paper by Crawford and Lewis and in the accompanying Editorial.

In spite of the well-known difficulty in extrapolating from one species to another, it would, in my opinion, have been irresponsible not to enjoin caution in the use during early pregnancy, of a drug known to be teratogenic in rats, until such time as an adequate quantity of human data showed the risk to be negligible. Happily, the clinical studies that have appeared since our book chapter[8] have now gone a long way towards showing that the risk appears to be negligible following the type of anaesthetic which has been included in the various surveys. For this, we may all be thankful.

Finally, our caution was obviously directed towards the period of organogenesis, which extends to 16 weeks. A trimester is by definition 13 weeks (as it was interpreted by your Editorial writer) and, allowing for uncertainty over dates, we gave our view that nitrous oxide was contraindicated during the first two trimesters.[8]

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J.F. NUNN

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A reply

Dr Nunn's letter adds important information regarding the safety of nitrous oxide as an anaesthetic during pregnancy[1,2] which had not been published at the time of writing the Editorial that accompanied the article by Crawford and Lewis. He also correctly points out that Amos *et al.*[3] have reported that DNA synthesis, as assessed with the deoxyuridine suppression test, was abnormal in a group of surgical patients admitted to the intensive care unit. These patients had been exposed to nitrous oxide for 1–6 hours. It was an oversight not to mention the fact that nitrous oxide is a weak teratogen in rats; this was assumed to be common knowledge.[4]

Dr Nunn's many contributions in this field of investigation have been at the cutting edge of scientific endeavour. Thus, it is gratifying to know that he considers that recent clinical studies have gone a long way toward demonstrating the safety of nitrous oxide when administered to pregnant patients who undergo operations.

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Morphine-induced respiratory depression in the elderly

In a recent study by Daykin *et al.* (*Anaesthesia* 1986; **41:** 910–4) which compared the acute respiratory effects of morphine on young and elderly patients, the authors stated 'the acute respiratory depression 10 minutes after a single intravenous injection of morphine is similar in elderly and young subjects.' Their aggregate statistics do seem to justify this conclusion, but I think there is more information to be gleaned from their results.

First, a simple inspection of their Fig. 1 shows that five of 12 elderly subjects had rather large decreases in the slope of their carbon dioxide response curves; yet only one of the 12 younger subjects experienced a similarly large decrease in slope. As nearly as I could calculate, the average decrease in slope for these five elderly subjects was almost three times the average decrease for all subjects.

Second, the $\dot{V}_{7.3}$ results, although not statistically significant, show the elderly group to have a 40% greater reduction in ventilation than the younger group (39.3% *versus* 28.1%) and a considerably larger intragroup variation. In fact, a rough power estimate is 0.4, meaning that the probability of a type II error was approximately 60%. In other words, there is a good chance that a real difference was not detected, either by chance or because the study group was too small.

Resting ventilatory measurements can be misleading because of the curve at the lower end of the carbon dioxide response curve. The carbon dioxide response curves are more important: they relate directly to the patient's sensitivity and responsiveness to increasing levels of carbon dioxide due to whatever cause (airway obstruction, sleep, increased carbon dioxide production, and so on).

My point is that the author's conclusions seem incomplete. They should have emphasised more strongly the greater variability in acute respiratory response to morphine in elderly patients indicated by their study, and stressed the importance of careful monitoring when narcotics are administered to older patients.

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D.L. Bourke

A reply

Thank you for allowing us to reply. Dr Bourke makes two statistical points. In the first, he rightly states that

Table 1. $\dot{V}_{7.3}$ and resting $P\mathrm{E}'\mathrm{CO}_2$ data.

	Control		Morphine	
Age group	Mean (SEM)	95% Confidence limits	Mean (SEM)	95% Confidence limits
$\dot{V}_{7.3}$ *(litres/minute)*				
Young	28.5 (2.22)	23.6–33.4	20.5 (1.98)	16.1–24.9
Old	33.4 (3.34)	26.0–40.8	20.2 (2.60)	14.5–25.9
Resting $P\mathrm{E}'\mathrm{CO}_2$ *(kPa)*				
Young	5.0 (0.08)	4.82–5.18	5.2 (0.15)	4.87–5.53
Old	5.0 (0.14)	4.69–5.31	5.3 (0.11)	5.06–5.54

the slope of the carbon dioxide response curve was noticeably decreased in five elderly subjects but in only one young subject. Statistically (Fisher's exact test, including extreme possibilities) this is not significant, but the relatively small trial numbers cannot exclude a sub-set of the elderly population who responded differently from the rest. The second statistical point considers the possibility of a type II error in the $\dot{V}_{7.3}$ data. We suggest that Dr Bourke's rough power estimate of 0.4 is unnecessarily low but, again, we accept that the relatively small number of subjects in our study does not exclude the possibility of a type II error in our results.[1,2] In order to demonstrate the spread of the data in our trial we append Table 1, which shows the 95% confidence limits for $\dot{V}_{7.3}$ and resting $P\mathrm{E}'\mathrm{CO}_2$.

Dr Bourke's final paragraph is puzzling because we thought we had stressed the limitations of our findings when the clinical use of morphine is to be considered. If we did not, then we apologise. However, we would point out that our final sentence was 'there is no evidence, however, to alter the view that it is wise to use minimal doses and exercise extra care in monitoring older patients given opiates.'

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D.J. Bowen
A.P. Daykin
D.A. Saunders
J. Norman

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For a discussion of sample size and type II errors we refer readers to:

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Training in fibreoptic techniques

Drs Davies and Hollway's letter (*Anaesthesia* 1986; **4**: 1265) was interesting; many anaesthetists have indeed little experience in fibreoptic tracheal intubation. This is likely to be due to lack of training in such procedures, the cost of a fibreoptic laryngoscope, or the unavailability of a bronchoscope.

We embarked on a stepwise training programme in fibreoptic intubation. In view of the cost and delicacy of the equipment, and difficulty in orientation in the use of such instruments, trainees first undergo a familiarisation and manipulation period. Then, intubation is attempted on models used for training in resuscitation. When they are successful at this, they start exposure of laryngeal structures of patients who are recovering from anaesthesia, using a nasopharyngeal approach after local anaesthesia of the upper airway. Thereafter, *oral* intubation is attempted in patients undergoing general anaesthesia as described by Davies and Hollway. We prefer to include glycopyrronium as part of our premedication, to reduce the amount of secretions and thus improve conditions. We have found that the oral approach for laryngeal exposure is easier than the nasal one. It is likely to cause less trauma, bleeding or smearing of the laryngoscope tip by lubricants from the nasotracheal tube. These are likely to obscure the view through the scope and present considerable difficulties to trainees. Therefore, the use of the nasal route is reserved until oral intubation is mastered by the trainees. If the former is used, a well-lubricated tracheal tube is inserted and positioned close to the larynx. The tube is attached to a swivel connector and a catheter mount which is connected to a Bain breathing system. The scope is passed through the suction part of the swivel connector, as its passage through the nose unprotected, may damage its tip. The use of this arrangement ensures an adequate level of anaesthesia and oxygenation during manipulation of the laryngoscope. Awake intubations are attempted when trainees have successfully intubated 30 anaesthetised patients. A viewing attachment is useful.

The value of fibreoptic laryngoscopy in difficult intubation is well recognised; its use to confirm the position of tracheal and bronchial tubes has been reported, and it has also helped us to diagnose the cause of obstruction of two tracheostomy tubes. Its wider use should be encouraged.

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Bradycardia after propofol infusion

We would like to report a case of a 66-year-old man who was anaesthetised for excision of a recurrent tumour of the posterior one-third of his tongue. He had had previous general anaesthetics which were uneventful.

The patient was in much pain from his recurrence and had received methadone 5 mg b.d. but no other treatment. Two weeks previously he had been examined without sedation in a dental chair, using a lignocaine topical spray; during this examination he had a syncopal attack followed by cardiorespiratory arrest. He was resuscitated successfully by being laid flat and ventilated by hand with 100% oxygen. He was later observed in the coronary care unit and no evidence of myocardial infarction or further dysrhythmia was discovered. Pre-operatively the patient had a regular pulse rate of 80 beats/minute, sinus rhythm, arterial blood pressure 170/80 mmHg, normal heart sounds and clear lung fields.

The patient was premedicated with temazepam 20 mg and oral atropine 1.0 mg. Anaesthesia was induced with propofol 2 mg/kg after pre-oxygenation and tracheal intubation facilitated with suxamethonium 75 mg. Anaesthesia was maintained with fentanyl 250 μg and a propofol infusion at 70 μg/kg/minute to a total of 940 mg over 4 hours. Further neuromuscular blockade was achieved with a bolus of atracurium 30 mg and maintained by an atracurium infusion at 0.4 mg/kg/hour.

Once he was settled on the operating table the patient had a pulse rate of 75 beats/minute, sinus rhythm and blood pressure 120/75 mmHg. The patient's pulse rate gradually decreased as anaesthesia progressed until, at the end of surgery, the rate was 50 beats/minute.

The atracurium infusion was stopped and the propofol infusion continued to provide sedation for transfer to the intensive therapy unit. The pulse rate continued to decline and was 40 beats/minute but in sinus rhythm, the arterial blood pressure was 110/70 mmHg but, as the patient's cardiac output was judged to be low, atropine 1.8 mg was given intravenously in divided doses; the pulse rate increased to 45 beats/minute. Isoprenaline 5 μg was given by slow intravenous injection, again without improvement in heart rate. Neuromuscular blockade was reversed satisfactorily with neostigmine 2.5 mg and atropine 1.2 mg. The propofol infusion was switched off and the patient allowed to breathe humidified 40% oxygen via the tracheal tube. The pulse rate increased to 62 beats/minute over the next 2 hours, and the blood pressure to 140/80 mmHg; the patient regained consciousness and his trachea was extubated.

This persistent postoperative sinus bradycardia is, in our experience, unusual after this form of surgery, although it is possible that some surgical interference with baroreceptors could be responsible. However, if this were the cause, a response to atropine would have been expected. Other possible causes include primary myocardial disease; however, both pre- and post-operative ECG were normal. Bradycardia during the administration of atracurium, has been described, but this does not persist long into the recovery period. Another possibility is that the bradycardia may be a result of the propofol infusion. There are little data yet on the cardiovascular effects of prolonged propofol infusion for maintenance of anaesthesia; however, cardiovascular effects have been investigated for a single dose of propofol, and one feature of some of these studies is the absence of a compensatory tachycardia in the presence of hypotension due to a reduction in systemic vascular resistance.[1] This does suggest that propofol does have some effect on heart rate.

We have no clear evidence of a direct relationship between the bradycardia and the propofol infusion in this patient but we believe there is some evidence to suggest an association. Have others made a similar observation?

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Reference

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AIDS and anaesthesia

We read the review by K.G. Lee and N. Soni (*Anaesthesia* 1986; **41**: 1011–6) with interest. We wish to add pain clinics to the fields listed as sites of contact between anaesthetists and patients who suffer from AIDS, and give an example below.

In these patients, opportunistic infections are not confined to the chest. A young man with proven HTLV III infection was recently admitted to our institution with severe intractable headache from cryptococcal meningitis. This symptom had proven refractory to all analgesics except parenteral pethidine, and the pain management unit was approached for advice on therapy. In view of the patient's history of drug abuse, the hazards associated with contaminated syringes and needles, his rapidly escalating pethidine requirement and presumed norpethidine cumulation, we changed his medication to oral methadone. Three months later he still has generally good pain relief and his current methadone dose is 100 mg/day. Our routine practice is to monitor plasma drug concentrations in patients on long-term opioid therapy but we have decided not to expose our technical support staff to the risks from handling blood from patients with AIDS.

Anaesthetists in *all* branches of the specialty need to be aware of the implications of treating these unfortunate patients.

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H. Owen
P.B. Gaukroger
D.A. Cherry
G.K. Gourlay

A comment in reply

As the incidence of HIV in the community increases, the areas of practice where anaesthetists' exposure to this virus may occur, will increase. Recent examples include a patient with burns who was found to be HIV antibody positive. The fairly haemorrhagic procedures associated with the management in this instance, pose potential problems for both anaesthetist and surgeon. Similarly, there are now obstetric patients who are HIV antibody positive and the question of the suitability of epidural analgesia is raised. Obviously, paediatric anaesthesia will also be affected. Re-evaluation of customary technique will be necessary in all these areas.

Dr Owen and his colleagues illustrate well two basic approaches to the management of these problems. Firstly, the avoidance of parenteral drugs where possible and, secondly, the rationalisation of what are considered essential tests to reduce the risk to medical and technical staff. The use of alternative, non invasive techniques should also be encouraged. For example, oximetry may reduce the need for arterial blood gas sampling in the management of respiratory problems in HIV antibody positive patients.

Rationalisation of technique, common sense and sensible precautions will significantly reduce the risk to anaesthetists. We agree fully that anaesthetists in all areas of the specialty should be aware of the implications of treating these patients but they should also be aware of the undiagnosed, or not yet seropositive, patient who they may treat. Anaesthetists are in contact with blood and saliva[1] frequently in normal daily practice and, consequently, are at risk. Simple precautions in normal practice, for example wearing gloves, may be sensible if the prevalence of the virus increases.

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Reference

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Oxygenation during electroconvulsive therapy

Drs Lew, Eastley and Hanning (*Anaesthesia* 1986; **41:** 1092–7) suggest manual ventilation with 50–60% oxygen during the convulsion as a means to prevent arterial desaturation. It was surprising to read that the use of 100% oxygen, either from a Boyle machine or from a cylinder with, for example, a Mapleson C system, was dismissed as unrepresentative of practice in this country. Perhaps its availability and use before the shock is given, might also be a means to avoid desaturation. There may then be no need to apply positive pressure ventilation during the convulsion, particularly if the airway is kept clear and the mask reapplied during this stage. Apnoeic oxygenation usually works quite well!

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D.W. Wood

Lew, Eastley and Hanning (*Anaesthesia* 1986; **41:** 1092–7) conclude from their study, that patients who undergo electroconvulsive therapy should be ventilated with an oxygen-enriched gas mixture during the convulsion. This recommendation is made on the basis of hypoxaemia detected in some patients ventilated with 40–50% oxygen before, but not during, the convulsion.

Ventilation of the lungs during the convulsion phase of ECT is often difficult (depending upon the degree of relaxation attained) and there is a real possibility of inflation of the stomach, with consequent risk of regurgitation. Had the authors ventilated with 100% oxygen instead of 40–50%, it is probable that the five cases of hypoxaemia would have been avoided. The authors defend their choice of oxygen concentration by the assertion that 'facilities for giving 100% oxygen may not readily be available'. Provided that a source of oxygen is available it is always possible to give 100% oxygen by using a Mapleson C system. A degree of rebreathing is probably irrelevant, in view of the brevity of the period of ventilation.

The authors should consider an investigation of the use of 100% oxygen prior to the shock before they make a general recommendation that ventilation should be attempted during the convulsion.

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R. Sneyd

A reply

The method of oxygenation with a self-inflating bag chosen for our study of oxygenation during ECT was that used in our hospital and, we believe, is commonly used elsewhere. Practice differs widely; inspired oxygen concentrations that ranged from 100 to 21% were noted in a recent survey.[1]

We cannot refute Dr Sneyd or Dr Wood's contention that 100% oxygen administered prior to the shock, would have prevented the hypoxaemia we observed. However, we find it doubtful that the small increase in oxygen stores that would result, would be adequate to prevent hypoxaemia in all patients, particularly if the convulsion is prolonged or a second shock is required. Oxygen consumption is markedly elevated during a convulsion, in contrast to tracheal intubation and other circumstances where apneoic oxygenation is used, when oxygen consumption is generally reduced. Pre-oxygenation did not influence the occurrence of cardiac dysrhythmias in one early study.[2]

We have experienced no difficulty with manual ventilation during the convulsion and continue to advocate its use during ECT to maintain the delivery of oxygen during the period of increased oxygen demand.

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R. EASTLEY

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Non vagolytic anaesthetic sequence—sinus arrest

We should like to clarify the points raised by Eastley, Davis and Strunin in their letter (*Anaesthesia* 1986; **41:** 1156–7) which referred to our reports of sinus arrest associated with the new neuromuscular blocking agents (*Anaesthesia* 1986; **41:** 89–90). Our reports were within a letter, so they were not comprehensive and much detail was omitted. They were, however, reported in the belief that the two episodes of sinus arrest would not have occurred had any relaxant other than vecuronium (or atracurium) been used, and that the obvious causes of dysrhythmias (hypoxia, hypercarbia, catecholamines or excess halothane) made no contribution.

On the specific points they raise, the halothane concentration was 0.5–1%, predominantly 0.5%, and cardiovascular variables before arrest gave no evidence of excess halothane. Ventilation was adequate in both cases and hypoxia was never apparent in either case. The 'British practice' to which they refer, of induction without the benefit of ECG monitoring, does not occur in this particular corner of Scotland; routine ECG monitoring in the anaesthetic room pre-dated these cases by several years.

With regard to the possibility that suxamethonium caused the dysrhythmias, suxamethonium is routinely flushed in with thiopentone in order to avoid not only the vagal effect of the second dose, but also respiratory embarrassment which could follow if 0.3 ml, 15 mg suxamethonium was inadvertently flushed in the recovery area. In any case, vecuronium was injected into a running drip through intravenous cannulae and there was no question of a second-dose suxamethonium effect. Concerning their observation that the port deadspace of a Venflon cannula may contain 0.1 ml, it is certainly possible, with some dexterity, to leave this deadspace full of liquid; however, in our hands, this is almost totally extruded when another syringe is attached.

Eastley, Davis and Strunin also comment on the lack of reports from Britain of suxamethonium dysrhythmias and assume this may be associated with a dearth of anaesthetic room ECG monitoring in Britain. ECG monitoring is not essential for detection of either rate change or rhythm irregularity; a finger (on the pulse) is still a diagnostic instrument in Britain. In addition, there is an abundance of literature about suxamethonium dysrhythmias, its acetylcholine-like effects and the pharmacology of its autonomic action.[1–6]

Finally, our original letter was not intended to make a case against the new neuromuscular blockers but to warn, as stated in our previous letter, other users of a common anaesthetic technique and to allow others to decide on the advisability of an anticholinergic agent for prophylaxis.

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J.M. THORP
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Epidural anaesthesia in the presence of a cerebral tumour

We read with interest the case report by Goroszeniuk and his colleagues, that concerned the management of labour using continuous epidural analgesia in a patient with a malignant cerebral tumour (*Anaesthesia* 1986; **41:** 1128–9). Their patient had evidence of raised intracranial pressure at the time her lumbar epidural was sited.

We have recently been involved in the management of a similar case. A 23-year-old primigravid Asian woman had an inoperable frontal glioma diagnosed during the 34th week of pregnancy.

We discussed the management of her labour at length with her obstetrician and her neurologist. Our intention was to provide the safest mode of delivery for both the mother and her baby. We decided to deliver our patient by elective Caesarean section under general anaesthesia with the judicious use of opiate analgesia postoperatively. This was performed uneventfully at the 39th week of pregnancy. The mother and her baby were discharged home on the 12th day post partum. The mother enjoyed 4 weeks of nursing her male infant with her family before she died.

We decided against epidural analgesia in this case. The incidence of accidental dural tap performed whilst an epidural is sited, decreases with the experience of the operator.[1] However, it remains an unpredictable hazard of siting an epidural. Lumbar puncture, performed in patients with raised intracranial pressure using a spinal needle, leads to a rapid clinical deterioration and is often fatal.[2–4] An inadvertent dural tap in a patient with raised intracranial pressure, using the larger Tuohy needle, could lead to immediate coning. Neurosurgical interventions, even in those patients with operable cerebral tumours, would be unlikely to salvage this situation.

We suggest that the presence of raised intracranial pressure should be a contraindication to the siting of an epidural. We feel that elective Caesarean section, under general anaesthesia, was the safest mode of delivery for our patient who had raised intracranial pressure secondary to her cerebral tumour.

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J.C. Sugden

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Dr T. Goroszeniuk and colleagues (*Anaesthesia* 1986; **41:** 1128–9) considered that regional anesthesia was appropriate in their patient with raised intracranial pressure during labour. In addition to their comments, two further points warrant consideration.

Firstly, they quote the incidence of inadvertent dural puncture as being very low in experienced hands. Hodgkinson[1] reported a rate of 2.3%, and Moir and Thorburn[2] reported a rate between 2 and 3%. These results were obtained in an obstetric population. The incidence of inadvertent dural puncture cannot be estimated in a population with raised intracranial pressure, but it is likely to be higher and to have disastrous consequences.

Secondly, it has been shown that lumbar epidural injection of local anaesthetic, or saline, increases intracranial pressure and that the increase is greater if intracranial pressure is already increased.[3] The authors of this report recommended that extradural anaesthesia should be used with extreme caution in patients with reduced intracranial compliance, and should not be used at all in a patient with intracranial hypertension or a space-occupying lesion.

If these two points are taken into consideration, regional anaesthesia may be less appropriate than the other methods discussed for the management of this difficult situation.

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J. Kinsella

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Goroszeniuk *et al.* (*Anaesthesia* 1986; **41:** 1128–9) describe continuous epidural analgesia during labour in a patient with a malignant cerebral tumour.

Their patient developed increasingly severe headache, indicative of increased intracranial pressure. They attributed this increase in intracranial pressure to increases in central venous pressure and arterial pressure with each contraction of the uterus.

Hilt *et al.*[1] described dramatic increases in intracranial pressure in patients with intracranial hypertension, when given repeated top-ups of local anaesthetic solution through an epidural cannula inserted for pain relief.

The patient who presented to Goroszeniuk *et al.* was clearly a difficult management problem. They rightly warn of the hazards of general anesthesia in patients with raised intracranial pressure. However, mention should also be made of the potential problems of regional analgesia.[2,3]

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G.A. Francis

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A reply

We are most appreciative of the interest shown by Dr Kinsella, Dr Francis and by Dr Chater *et al.* in our report. Dr Kinsella takes issue with two points; the incidence of inadvertent dural puncture, and whether extradural anaesthesia is contraindicated in patients with raised intracranial pressure. On the first point, your correspondent appears to have misread, or misinterpreted, the communication of Moir and Thorburn. They state quite clearly that the incidence of inadverent dural puncture in experienced hands is 0.2%.[1] Secondly, Wildsmith, in an editorial comment[2] on the work of Hilt, Gramm and Link[3] (cited by your correspondent), states that the administration of extradural anaesthesia in a patient with a raised intracranial pressure . . . 'is, as in many situations, a matter of fine judgment in balancing the advantages and disadvantages of alternative techniques'. We would entirely support this point of view. Epidural anaesthesia enabled us to help our patient achieve her most heartfelt wish; to be awake during the delivery of her baby.

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A matter of vigilance

It was with concern that we read Dr Green's Editorial (*Anaesthesia* 1986: **41:** 129–30), and his subsequent reply to Dr Eames' letter (*Anaesthesia* 1986; **41:** 961–2).

We do not dispute that it is important to monitor physiological variables continuously during anaesthesia and the immediate postoperative period. However, we were dismayed to read the medicolegal view that it is not acceptable to delegate these duties, even for a short time, to qualified nursing or paramedical staff. Such a ruling would seem to negate their training. The majority of operating lists in District General Hospitals (DGHs) are serviced by one anaesthetist. If he/she is to follow Dr Green's advice to the letter and never leave an anaesthetised patient, even to visit the recovery area, it means that a second patient must not be induced until the first is ready to return to the ward. The resultant delays would necessitate the curtailment of operating schedules by up to 50%. As it is inconceivable that finance would be made available to double the number of anaesthetists, the consequent increase in waiting lists would prove disastrous in terms of morbidity and possibly mortality.

Dr Green reports that only four out of 150 patients were unattended by their anaesthetist at the time of their mishap, and does not state over what period of time his survey was conducted. Is it reasonable to condemn all our patients to extra time on already overlong waiting lists, in the hope of safeguarding such a small minority? We believe not, and wish to record total confidence in the ability of our trained operating department assistants and recovery nurses to perform their traditional duties safely.

Busy DGH anaesthetists do not work in an Utopian environment, but strive to benefit as many patients as possible with the resources available to them. Medicolegal pronouncements that fail to recognise this fact, and are afforded editorial status in a prestigious journal, do little to make a difficult job any easier.

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A.M. Barr
P.J. Brock
R.M. Hall
R.H. Jago
D.M. McWilliams
T.G.C. Smith
C.S. Waldmann
E. Young
M.E. Rimmer

A reply

The fact that ten anaesthetists from the Royal Berkshire Hospital fail to appreciate the message contained in my Editorial, seems to reflect on my ability to communicate.

It was not suggested that anaesthetists need never delegate responsibility to paramedical staff for short periods in order to see a patient in the recovery room or attend to a life-threatening emergency. It is the unnecessary and prolonged delegation which may lead the anaesthetist into serious medicolegal difficulty, should a mishap occur during his or her absence from the theatre.

There was no question of a medicolegal pronouncement in the Editorial; it was a reminder of the unnecessary risks which are occasionally taken by anaesthetists and the possible consequences of so doing.

I recently attended Court in defence of an anaesthetist who left a patient, with a nurse in charge, on three separate occasions during a 90-minute dental operation. On one of these occasions he anaesthetised another patient for a short procedure, in an adjacent theatre. The patient died subsequently of malignant hyperpyrexia and the father was determined to obtain a verdict of criminal negligence against the anaesthetist because of his inattention. We managed, with great difficulty, to achieve a verdict of misadventure but only on the grounds that the anaesthetist's absence from the theatre made no difference to the outcome.

We do have a difficult and responsible job as anaesthetists. We deserve all the help we can get and, it is to be hoped, by drawing attention to the risks we occasionally have to take, the authorities will help to provide that perfection few can afford.

The patient under anaesthesia certainly deserves the undivided attention of a trained anaesthetist.

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R. Green

Editor's note: This correspondence is now closed.

Epidural block for elective Caesarean section

The observations of Drs Crawford, Davies and Lewis (*Anaesthesia*, 1986; **41:** 1046) on the reduced frequency of blood tranfusion in their patients who received an epidural anaesthetic, compared with those who received a general anaesthetic for Caesarean section, were interesting but I do not agree with their statement that 'it is doubtful that the anaesthetists concerned were markedly reluctant to start a blood transfusion in an awake patient'.

Blood loss at Caesarean section is difficult to estimate and is subjective. This estimate will be influenced by several factors. An anaesthetist who expects that blood loss is reduced in Caesarean section under epidural, may underestimate the observed blood loss. He may overestimate the observed blood loss at a Caesarean section under general anaesthesia, particularly if he has used a volatile agent after delivery. Hypotension under general anaesthesia may be assumed to be due to hypovolaemia and treated with transfusion. Hypotension under epidural may be assumed to be due to sympathetic blockade and treated with ephedrine. Comments on blood loss and operative difficulty will be restrained if the patient is awake.

The anaesthetist may be reluctant to start a blood transfusion for other reasons. He has already volume loaded the patient with up to 2000 ml fluid and does not wish to over-transfuse the patient. The patient may regard the transfusion with alarm. The symptoms of a transfusion reaction, however minor, would be very difficult to treat in a patient undergoing surgery while awake.

Ultimately, his decision to transfuse will be influenced by what he believes is happening to oxygen transport and delivery to the brain. As long as the patient is awake, happy and responds to his conversation, he can rest assured that her circulation is adequate.

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Portsmouth, Hants.

A.M. Severn

A reply

Thank you for the opportunity to reply. We cannot agree with the opinions expressed by your correspondent.

Anticipation of relatively less blood loss when a section is conducted under an epidural is, we submit, very unlikely to influence the assessment of blood loss during the actual procedure.

Hypotension that occurred during the elective operation—the block, by definition, would have been established for at least 20–30 minutes before the uterus was incised—would certainly prompt the immediate suspicion of hypovolaemia due to haemorrhage. We would not expect a fall of blood pressure so sudden and severe as to prompt the administration of ephedrine, to be reflective of a sudden extension of the vasomotor block.

Our obstetricians' comments are no less restrained when general anaesthesia is administered, than when the mother is awake. In any case, they are aware of the fact that it is the anaesthetist's responsibility to gauge the extent of haemorrhage and to decide whether or not blood should be given, although we appreciate that they should tell us if the myometrium requires to relaxed further in order to facilitate a gentle delivery.

The volume of fluid used for preloading has no relevance to a decision about blood replacement. The preload simply satisfies the demand imposed by an increase in circulatory capacity.

An attentive and sympathetic anaesthetist should be able to explain reassuringly to the mother, why he considers it advisable that she be given a blood transfusion. Frankly, if the anaesthetist does not possess those characteristics, he/she should not have embarked upon the procedure in the first place; we are doctors, not technicians.

We have observed at least one transfusion reaction in this situation. It was treated promptly and successfully, without drama or complication. Indeed, it was the mother herself who, by reporting some tingling in her face, initiated the diagnosis.

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J.S. Crawford

Benefits of pulmonary artery catheterisation

It is a matter of regret that Dr J.C. Stoddart, in his book review (*Anaesthesia* 1986; **41:** 1167), attempts to perpetuate the myth that the use of pulmonary artery flotation cathethers 'has never been shown to improve the survival of critically ill patients'.

The editors of *Invasive Procedures in Critical Care*, published in 1985, may be forgiven this error, allowing for the usual delay of several years between the preparation of a contribution and its eventual appearance, but surely your reviewer has an obligation to draw to the readers' attention, any major error that time has brought to light.

In 1982 Shoemaker and colleagues[1] published evidence from a prospective trial, of the benefits in outcome to be gained by the use of pulmonary artery flotation catheters. Further data which confirm this finding, have been published by the same group,[2,3] whilst no-one has published a refutation of their methods or results.

Unless your reviewer has evidence to the contrary, which we would hope to see published, support for such negative statements should be much more guarded. Or is a mortality reduction from 29% to 4% of no interest?[3]

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R.G. Wilkins
J.D. Edwards

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A reply

Thank you for giving me the opportunity to see the letter from Drs Wilkins and Edwards.

I am sorry that they take exception to a book review. It is my opinion that, amongst a reviewer's duties, is the need to draw attention to interesting statements of opinion. The chapter to which I drew attention, was written by a man who has considerably more experience with the use of flotation cathethers than I, and I therefore felt his opinion was worth restating and I choose to stand by that opinion.

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J.C. Stoddart

Croup, nebulised adrenaline and preservatives

The case reported by Drs Remington and Meakin (*Anaesthesia* 1986; **41:** 923–6) illustrates the efficacy of nebulised adrenaline in the treatment of croup. This treatment is widely used in North America, where racemic adrenaline is the usual preparation employed. In our hospital we have prepared a solution of 1%

adrenaline especially for nebulisation, in the fear that the metabisulphite content of adrenaline 1 : 1000 BP might further damage the compromised respiratory tract.

The irritant nature of sulphur dioxide released from sodium metabisulphite is well known to home brewers. Inhalation of sulphur dioxide in concentrations as small as 6–12 p.p.m. causes irritation of the nose and throat, and bronchoconstriction.[1] Koepke and colleagues[2] showed that sulphur dioxide was evolved in significant amounts from bronchodilator solutions for nebulisation. The isoprenaline preparation they studied, often produced 6 p.p.m. of sulphur dioxide. This solution contained 1.5 mg sodium metabisulphite, whereas the 5 ml adrenaline 1 : 1000 administered to the patient in this report, contained 5 mg sodium metabisulphite.[3] Incidentally, Koepke and colleagues could detect only 0.2–0.4 p.p.m. sulphur dioxide in racemic adrenaline nebulised in the usual manner.

The 1% adrenaline for nebulisation used in this hospital was originally preserved with chlorbutol and chlorocresol but, at present, benzalkonium chloride is employed. It may be that these agents are also respiratory tract irritants, but there is less evidence that this is so. The shelf-life of this preparation is 6 months or less, so the pharmacy expends considerable effort on an agent that is used infrequently. Nevertheless, nebulised adrenaline does enable some children with severe croup to manage without intubation which usually leads to a long, hazardous and expensive period of intensive care.

We wholly support Drs Remington and Meakin in their encouragement of anaesthetists to use nebulised adrenaline in croup, but have reservations regarding their statement that 'nebulised adrenaline 1 in 1000 in a dose of 5 ml provides a safe and effective alternative to racemic adrenaline . . .', on account of the sulphur dioxide. We would be interested to know if large numbers of patients have been safely managed with this preparation. If so, adrenaline 1 : 1000 BP is clearly a more practical proposition for most hospitals.

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Cervical epidural analgesia during carotid artery surgery

Kainuma *et al.* (*Anaesthesia* 1986; **41:** 1020–3) state that cervical epidural analgesia provides a safe and reasonable technique for patients who undergo carotid artery surgery. How can this conclusion be so confidently drawn from a study that involved only three patients? Considering these, case 1 sustained a hemiparesis despite the intra-operative use of a temporary bypass shunt; case 2 was uneventful, although general anaesthesia was also maintained, except during the period of carotid clamping. Finally, the operation on case 3 was abandoned due to the development of signs of cerebral ischaemia following temporary carotid occlusion.

The authors assert that, under general anaesthesia, techniques to monitor the adequacy of cerebral perfusion during carotid artery surgery are either not very reliable, or are clinically unavailable. Neurophysiological monitoring of hemisphere function during temporary carotid occlusion has been shown, by a number of authors, to be a reliable indicator of ischaemia and the need for the insertion of a temporary bypass shunt.[1–3] The presence of a neurophysiologist is necessary for the interpretation of an electroencephalogram but the use of the cerebral function monitor, combined with stump pressure measurement, has been found to be an accurate and reliable predictor of inadequate cerebral perfusion, and this form of monitoring can readily be managed by the anaesthetist.[4]

Finally, cervical epidural analgesia is not without risk and it is disturbing that a technique which, among other risks, has the potential to cause arterial hypotension, should be recommended as safe for the management of patients who undergo carotid surgery. Many of these are hypertensive, with or without coincident ischaemic heart disease and, hence, can poorly withstand a significant fall in blood pressure. Kainuma *et al.* recommend sufficient cardiopulmonary monitoring but they do not state what methods were used in their patients.

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during carotid endarterectomy. With results of surgery and hemodynamics of cerebral ischemia. *Mayo Clinic Proceedings* 1981; **56:** 533–43.
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A reply

Thank you for giving us the opportunity to respond to this letter.

The major point which we wished to emphasise in our article was the outstanding reliability of assessment of patients' neurological states on carotid clamping during cervical epidural anaesthesia. Cases 1 and 3 had suffered from narrowing of bilateral carotid arteries in the pre-operative period and, therefore, we were anxious about the adequacy of cerebral blood perfusion on unilateral carotid clamping. Cervical epidural anaesthesia gave indisputable evidence of the need for a temporary bypass in cases 1 and 3. In cases 2 and 3, we performed tracheal intubation prophylactically for fear of respiratory depression and used general anaesthesia with nitrous oxide in oxygen, except during the period of carotid clamping. However, we recently anaesthetised three patients undergoing carotid endarterectomy, solely with cervical epidural anaesthesia without tracheal intubation, and confirmed its safety and reasonableness. In the near future, we intend to publish the results of this technique in a larger number of cases.

We do not deny the accuracy and reliability of neurophysiological monitoring. Sundt *et al.*[1] stated that the electroencephalogram (EEG) had proved a sensitive means of monitoring neurological function, and never had a patient emerge from anaesthesia with a new neurological deficit that was not predicted by the 16-channel EEG tracing. However, relatively few medical centres presently use the EEG because it is technically difficult and accurate interpretation requires skilled personnel.[2] Electrical disturbances from the electrocautery and other electrical apparatus in the operating theatre, may interfere with the EEG signal.[3] An interindividual relationship between regional cerebral blood flow and EEG mean frequency was denied by Trojaborg *et al.*[4] Moreover, volatile anaesthetics at increasing concentration lead to a progressive reduction in frequency and to periods of burst suppression followed by electrical silence, although isoflurane did not affect the ability to detect the onset of cerebral ischaemia.[5] These disadvantages often limit the use of the EEG as a monitor of cerebral function during anaesthesia for carotid surgery.

The cerebral function monitor (CFM) is a device that displays the power of the EEG signal from a single pair of electrodes after the signal has been filtered to give a frequency band of 2–15 Hz, and it eliminates a great deal of interference artefact. The use of the CFM combined with stump pressure measurement, may not require the personnel and equipment needed for a full-scale EEG system. However, according to Cucchiara *et al.*,[2] the CFM missed subtle EEG changes and borderline low regional cerebral blood flow in a few patients. Additionally, stump pressure does not necessarily reflect flow as long as resistance can change. Therefore, these measures are not absolutely reliable as monitors of a patient's neurological state on carotid clamping, even if the CFM is used in combination with stump pressure measurement.

We always place an intra-arterial cannula in a radial or dorsalis pedis artery before the cervical epidural catheter is inserted. Continuous monitoring of the systemic arterial pressure is required to treat arterial hypotension after epidural anaesthesia with lignocaine. Clinically, it is easy to maintain the systemic arterial pressure adequately by optimal volume titration and intravenous ephedrine. Central venous and/or pulmonary arterial pressures are measured, depending upon the patient's pre-operative condition. In addition, a pulse oximeter is used to measure oxyhaemoglobin saturations continuously. Continuous intra-arterial oxygen tension monitoring is sometimes performed. Many of the patients who undergo carotid endarterectomy have ischaemic heart disease, so we are always careful to avoid hypotension. Recently, Davis *et al.*[6] reported that, in dogs, cardiac sympathetic blockade with thoracic epidural anaesthesia could reduce the sympathoadrenal stress response to myocardial ischaemia and/or to the surgical preparation. It is, accordingly, possible that cervical epidural anaesthesia is appropriate for patients with ischaemic heart disease.

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Hypophosphataemia

We recently admitted to our intensive therapy unit a 38-year-old male with hepatitis type A and progressive encephalopathy, who had previously been well. He was jaundiced and drowsy; he responded to verbal commands but was poorly attentive. Initial investigations showed a normal biochemical and haematological profile; liver function was abnormal with a hepatocellular-type picture, and abnormal clotting times. Serum Ca^{2+} was 2.25 mmol/litre, and PO_4 0.27 mmol/litre.

Later that day his condition deteriorated and it was decided that tracheal intubation and controlled ventilation of his lungs were necessary. Neurologically, he was unresponsive to all stimuli, areflexive but with equally reactive pupils. No sedation was given other than that required for intubation. The phosphate level had decreased to 0.16 mmol/litre the following day, and intravenous replacement therapy of 30 mmol/24 hours was commenced. His neurological state was unchanged.

The serum phosphate was 0.29 mmol/litre on the 3rd day of admission and he received an additional 40 mmol dipotassium hydrogen phosphate. The level of coma persisted. His serum phosphate was unrecordable by the 4th day and a phosphate load of 120 mmol was administered. He remained deeply unconscious. However, he began to breathe spontaneously within the subsequent 24 hours and made efforts to reject the tracheal tube. The serum phosphate was 0.37 mmol/litre and a further dose of 90 mmol in 24 hours was administered. By the 5th day of admission, he was alert and orientated; arterial blood gas analysis was satisfactory so his trachea was extubated. Liver function began to improve and the phosphate increased to 0.92 mmol/litre. The serum phosphate was 1.23 mmol/litre on the 6th day, after a further load of 40 mmol in 24 hours. He was discharged to the general ward and went home 6 days later with near normal liver function.

Complications from severe hypophosphataemia include muscle weakness, acute respiratory failure and neurological abnormalities that include paraesthesiae, seizures, ataxias and coma.[1,2] Lentz[3] recommended parenteral doses of 0.08 mmol/kg/6 hours for acute uncomplicated hypophosphataemia, increased to 0.16 mmol/kg if prolonged and with multiple causes. The maximum rate of infusion thought to be safe is 0.24 mmol/kg in a 6-hour period. However, neither the response to therapy nor the extent of the deficiency can be predicted, because of the transcellular shifts that can occur with normal phosphorus stores but low serum phosphate. Therefore, the therapeutic guidelines must, of necessity, be empirical, and require close monitoring. In the face of a rapid decrease in serum phosphate, an intravenous load of 210 mmol over 48 hours was given, which is well in excess of the recommended regimen. Intravenous phosphate can cause hypocalcaemia, metastatic calcification, hypotension, hyperkalaemia, hypernatraemia and dehydration.[2]

Fortunately, none of these complications occurred and the patient made a rapid and uneventful recovery.

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An antipollution device for use with the Jackson–Rees modification of Ayre's T-piece

Several devices of varying complexity have been described for scavenging gases from paediatric breathing systems.[1–3] This is another.

A conical, invertable seal (similar to a diving drysuit seal) of 2 mm expanded neoprene is fashioned over the open end of a clear Perspex tube (internal diameter 19 mm). The tube is then attached to a standard Penlon male adaptor (Fig. 1). The seal accepts a Dennison valve and forms a gas-tight seal around its base. The valve setting is not affected by its insertion, which can be confirmed through the clear Perspex. The Penlon connector is plugged into a pas-

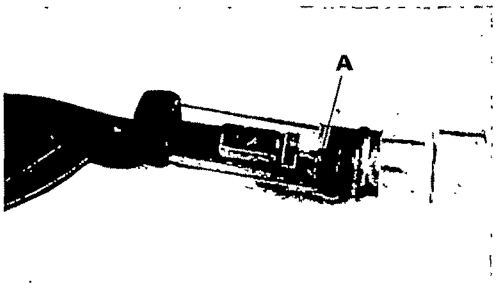

Fig. 1.

sive scavenging system. The total resistance of the system (over the flow range 1–20 litres/minute) is not increased by the addition of the device. At clinical flow rates of up to 8 litres/minute the pressure drop is around 0.02 kPa. The device does not interfere with assisted ventilation by hand.

In over 4 months at the Royal Hospital for Sick Children, Edinburgh, no problems have emerged from its use. The device could be modified for use with active scavenging systems by the addition of a large air inlet at A.

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D.L. Paul

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Caudal epidurals—a further caution

Drs Watt and Huggins (*Anaesthesia* 1986; **41:** 1157–8) presented an interesting case of a caudal epidural. Diminution in the capacity of the vertebral canal due to the movement of the body, was shown nearly a hundred years ago.[1] Repeated straight leg raising, by altering the size of the spinal canal, should cause changes in intrathecal pressure and would account for the extensive spread of local anaesthetic in the epidural space.

However, I was surprised that they used rapid intravenous infusion of 6.5 litres of fluid to treat hypotension: judicious use of a vasopressor would, in my opinion, have been the logical treatment. Avoidance of a massive infusion would have prevented diuresis, urinary retention and catheterisation.

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Reference

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Caudal epidural—possible subdural spread?

A picture of excessive spread with delayed onset and unexpectedly profound blockade following caudal administration of 40 ml 0.25% bupivacaine, is characteristic of subdural extra-arachnoid injection.[1–3] Is it possible that, in the case described by Drs Watt and Huggins (*Anaesthesia* 1986; **41:** 1157–8) the dura, but not the arachnoid, extended to within their needle's length of the sacral hiatus?

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Safety of a new system for continuous airway pressure

The report of an investigation into the safety of the Downs CPAP system by Jenkins and Van-Besouw was interesting (*Anaesthesia* 1986; **41:** 1155–6). We too have been worried by the absence of a blow-off valve in the system.

Jenkins and Van-Besouw showed that the system blows-off at a pressure of 5.3–6.4 kPa, depending on flow, and conclude therefore that it can be safely recommended. We contend that a system that can apply a continuous pressure three times greater than that used for CPAP, cannot be considered safe. Patients in the intensive care unit, may be at particular risk from pulmonary barotrauma, or the effects of raised intrathoracic pressure on the cardiovascular system. Elimination of the possibility of patients' exposure to such high airway pressures is required.

We have considered this problem and suggest that the system be modified as follows. A 22-mm T-junction with a CPAP valve on the side-arm is interposed between the air-entraining Venturi device and the patient system. The pressure setting of this second valve is arranged to be 0.5 kPa greater than the setting of the distal CPAP valve. Thus, if the distal valve becomes blocked, or the system obstructed for any reason, the proximal valve will act as a blow-off valve and prevent the patient from exposure to potentially dangerous airway pressures.

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Kinked tube or bronchospasm?

In the attempt to distinguish between the wheeze of bronchospasm and that of a kinked tracheal tube, the following observation is often useful.

Gas which flows at a sufficient rate through a large number of small tubes gives rise to a myriad of individual musical sounds; in contrast, a partly obstructed tube tends to produce only one basic root tone. Any musical tone is composed of a number of harmonics, but this does not obscure the sensation of a single voice imparted by the kinked-tube sound.

The second difference between the two sounds lies in their quality, since high-frequency noise or hiss is a much more obvious component in the kinked-tube airflow disturbance, than in that which occurs in bronchospasm.

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D.G. Ross

All that wheezes is not asthma—yet again

This is a note in support of Dr Mackenzie's statement (*Anaesthesia* 1986; **41**: 1063–4), that the occurrence of bronchospasm in the middle of an anaesthetic should always raise the suspicion of a mechanical problem.

A 70-year-old woman (height 1.61 m, weight 60 kg) was anaesthetised in order to receive an aortic valve prosthesis. Ventilation of the lungs was without incident at first (Fig. 1(a)). Towards the end of cardiopulmonary bypass the surgeon complained that both lungs were overdistended. The ventilator setting (Servo 900 B) could not be increased to a normal volume with the volume control without the airway pressure exceeding the present limit of 4.5 kPa (Fig. 1 (b)). This tracing shows a constant expiratory flow rate typical of mechanical obstruction. The airway pressure rises steeply, suggestive of increased airway resistance

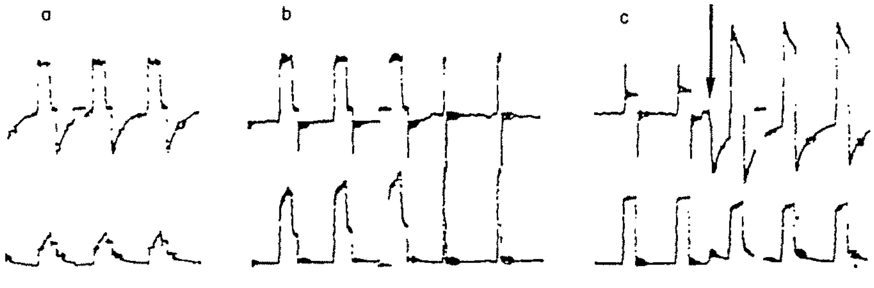

Fig. 1. Airway flow and pressure. (a) Before cardiopulmonary bypass; normal pattern of pressure increase during inspiration, normal expiratory flow. (b) Three completed breaths during obstruction, followed by two interrupted as the airway pressure exceeds the present limit; abnormally rapid increase in airway pressure at each inspiration, abnormal square-wave expiratory flow, typical of obstruction. (c) Now pressure controlled, there is very little flow in relation to the inflation pressure; expiratory flow is still abnormal (first three breaths). The arrow denotes replacement of the tube, immediately followed by a normal expiration, as inspiratory and expiratory flow are now unhindered.

which lessens as inflation proceeds.[1] Manual ventilation confirmed the increased inspiratory resistance, with very poor expiratory flow. The differential diagnosis was acute airway obstruction or asthma. In order to achieve some ventilation at reasonable airway pressures whilst a strategy was worked out, the ventilatory working pressure was reduced to 3.5 kPa and the present minute volume increased greatly in order to produce pressure-controlled ventilation (Fig. 1(c), *left*). This shows a square-wave pressure pattern, with a rapid initial flow spike followed by low inspiratory flow. Expiratory flow rate was constant, as before. The position of the tracheal tube (Portex, 8 mm) was now checked. It had initially been placed with the 22-cm mark at the gums, but was now 2–3 cm further out. The cuff, which had not been overinflated, was deflated, the tube repositioned at 22 cm and the cuff reinflated. Figure 1(c) shows the immediate improvement in flow, at the arrow.

Presumably, cranial displacement of the tube allowed the intratracheal part to curve, so that the distal opening lay against the tracheal wall and produced the flow and pressure patterns described above. Bronchial intubation is not the only cause of airway obstruction in intubated patients! The presence of centimetre markings on the tube and recognition of the abnormal airway pressure and flow pattern, enabled the correct diagnosis to be made.

University Hospital,
S-221 85 Lund,
Sweden

R. FLETCHER

Reference

1. FLETCHER R, NORDSTRÖM L. The effects on gas exchange of a large mediastinal tumour. *Anaesthesia* 1986; **41:** 1135–8.

Nasotracheal intubation: a problem with another approach

We read Dr P.J. Wright's letter (*Anaesthesia* 1986; **41:** 1057–8) with some interest.

The technique described has been used in this intensive therapy unit for several years, although we claim no originality. The strategy not only protects the cuff of the tube and the nasal mucosa from damage, but it also prevents internal soiling of the tube.

However, we consider that this approach is not entirely without danger and in a recent case, the glove finger was lost.

A 49-year-old woman with an acute exacerbation of bronchial asthma was transferred to the intensive therapy unit for mechanical ventilation of her lungs. She was blue and exhausted on arrival. The airway was rapidly assured with an orotracheal cuffed tube and positive pressure ventilation of her lungs started. It is our practice when patients require only a few hours of artificial ventilation, to re-intubate via the nasal route using a tube of internal diameter 8.5 mm or larger: this relatively large size is chosen in order to allow fibreoptic bronchoscopy to be undertaken.

A Portex 8.5 mm low-pressure, high-volume (Profile) cuffed tracheal tube, with the protective glove finger in place, was inserted into the right nostril. It did not prove possible to pass the tube into the oropharynx, despite repeated manipulation. However, only moderate pressure was used to push the tube forward. The tube was then withdrawn so that it could be re-inserted via the left nostril. The glove finger was found to be missing from the tube. We therefore explored the possible sites of lodgement. The nasal cavity and postnasal space were thoroughly examined by ourselves and subsequently by a consultant ENT surgeon. Fibreoptic bronchoscopy and gastroscopy were also undertaken to rule out its presence in the tracheobronchial tree and stomach, respectively. The glove finger was not found.

The only conclusion was that the glove finger was pushed under the nasopharyngeal mucosa, although the applied pressure was only moderate. An X ray of the sinuses/skull did not, as we expected, show anything of note, although we are aware that the glove finger is radiolucent.

The patient and her relatives were fully informed of the problem, and we await developments. So far (3 months after the event) there has been no sign of the glove finger or any evidence of nasal, sinus or retropharyngeal infection.

We have since considered a number of simple techniques to ensure that we do not lose another glove finger, but have so far not arrived at a satisfactory and practical solution. We believe that there is an American 'umbrella-like' device which may be inserted down a tracheal tube to emerge as a protective, but we do not think that it is marketed in the United Kingdom.

South Cleveland Hospital,
Middlesborough,
Cleveland TS4 3BW

M.D. KUWELKER
P.G. LAWLER

Anaesthesia and the subclavian steal syndrome

Dr Thompson (*Anaesthesia* 1986; **41:** 1026–8) may well have understated the significance of this observation, that hyperventilation can cause the reappearance of the radial pulse in subclavian steal syndrome. It seems, to me, that he has described a model that illustrates the inadvisability of hyperventilation in many situations in anaesthesia. The increase in cerebral vascular resistance caused by reduction in $Paco_2$ will occur regardless of the patency of the proximal subclavian artery. That this can cause a reversal of flow in the vertebral artery in this syndrome, does not imply that these patients are more at risk of cerebral ischaemia than a comparable patient with a patent subclavian artery, merely that it is more easily detected (or rather, inferred).

Thus, I regard this report as a further argument in favour of routine monitoring of end tidal carbon dioxide in all patients in whom a reduction in cerebral blood flow may be deleterious. This would include all patients known to have cerebral vascular disease and, perhaps, all those at risk of postoperative confusion, that is, those over a certain (arbitrary) age.

National Heart Hospital,
London W2
M.S. READ

Difficult extubation due to a kinked pilot tube

The letter from Drs Tanski and James (*Anaesthesia* 1986; **41:** 1060) was interesting. They draw our attention to a hazard which may occur with Mallinckrodt tracheal tubes. The physical characteristics of tracheal tubes with large-volume, low-pressure cuffs have recently been studied. All these tubes, produced by various manufacturers and available in the United Kingdom, are described as for oral or nasal use (Table 1). In my view, it is unlikely for the pilot tube situated in the wall of the tracheal tube to be kinked by the retaining bandage. However, this complication may have occurred, since the pilot balloon on the Mallinckrodt tube gives no warning of the failure of the cuff. The pilot balloon, when deflated, should lie flat and should be so constructed as to give adequate indication of the inflation of the cuff.[1]

Table 1. Physical characteristics of large-volume, low-pressure cuffed tracheal tubes.

Tube type (size 8)	Distance to take-off point of pilot tube (cm)	Shape of pilot balloon
Portex, *Profile*	17	Flat
Franklin, *Sensive*	18.3	Flat
Mallinckrodt, *Hi-Lo*	20.5	Cylindrical
Rusch, low-pressure	23.2	Flat
Shirley, low-pressure	20.5	Flat
Warne, low-pressure	20.5	Flat

The pilot balloon on the Mallinckrodt tube is cylindrical in shape and gives no indication of the inflation of the cuff within the intracuff pressure range of 0–6.0 kPa. The balloon collapses only when the intracuff pressure is subatmospheric. Any patient who is intubated with this type of tube and breathing spontaneously, is potentially at risk and exposed to the danger of aspiration unless the cuff is kept inflated with a pressure gauge in line.

Victoria Hospital,
Thursby Road,
Burnley BB10 3HP
S. MEHTA

Reference

1. BSI. *Specification for endotracheal tubes. BS3487.* London: British Standards Institution, 1962.

Children and explosive anaesthetics

The letter on the above topic from J.P. Barcroft *et al.* (*Anaesthesia* 1986; **41:** 1062–3) is interesting and concludes with a question about the safety (or otherwise) of the continued use of explosive anaesthetics at the present time.

As the authors of that letter observe, 'in case of subsequent explosion any claim for damages would be indefensible', and most departments have become unhappy about the use of explosive anaesthetic agents in the operating room. The introduction of ketamine and the ease of its injection, even intramuscularly, have made the use of flammable and explosive agents much less necessary; it is the method of choice in our institutions. Other, similar agents are also (and will, no doubt, become) available.

Caritas Medical Centre,
Shamshuipo,
Kowloon, Hong Kong
Z. LETT

Anaesthesia in difficult circumstances

We are lecturers in anaesthesia at the University Teaching Hospital in Zambia and, at present, have many shortages in our anaesthetic drugs and equipment. We have developed two techniques which may be of interest to colleagues in similar circumstances in other parts of the world.

Central venous pressure (CVP) measurement. We have no manometer sets for the estimation of CVP, so we piggyback a butterfly onto the hub of the giving set connected to the CVP cannula. We measure the CVP using this column of fluid, and control the system by the clamp of the drip set (we have no three-way taps, so this also provides a separate route for infusion).

Local anaesthesic gel for tracheal tubes. We have found that, in the absence of proprietary preparations of local anaesthetics for this purpose, 1 ml 10% lignocaine mixed with 9 ml KY jelly makes a very suitable anaesthetic lubricant for the tracheal tube, where this is indicated. Lignocaine 4% is also suitable.

University Teaching Hospital,
Private Bag RWI,
Lusaka, Zambia

I.H. Wilson
J.R. Sinclair

Deviations from international standards

The letter from Dr Kumar *et al.* (*Anaesthesia* 1986; **41:** 1055–6) is interesting. This highlights the fact that safety standards are far from satisfactory in several countries. In addition to the deviations from international colour codes as described, the Boyle anaesthetic machine used by them probably did not contain a pin-index system which could have prevented the accident.

During my visit to Guyana I came across a 'Boyle major' anaesthetic apparatus which was a potential source of accidents that could result from wrong cylinder connexions. The anaesthetic machine had all the features of the Boyle major except that the cylinder yokes did not contain the pin-index system. The pin-index system was introduced into practice in 1955 and the Boyle major anaesthetic machine was manufactured several years later.[1] Therefore, one would not expect to find a Boyle major without the pin-index system. There are limitations to the enforcement of safety standards in these countries, as there are no reliable agencies to deal with irregularities. The theatre may be equipped with familiar brands and models of equipment but they may not have the same standards and safety features as one would expect to find in the developed countries, where the enforcement of the safety regulations is strict.

Anaesthetists from the overseas who are willing to serve in these countries, should be aware of these potential problems.

Queen Elizabeth Hospital,
University of West Indies,
Barbados, West Indies

K.B. Shankar

Reference

1. Watt OM. The evolution of Boyle apparatus, 1917–1967. *Anaesthesia* 1968; **23:** 103–18.

Halothane specific antibody

If hepatitis is suspected in any patient, anaesthetists are invited to send to this unit a sample of serum for testing. A sample of 10–15 ml blood should be taken and serum stored at minus 20°C. The sample should be sent by first class post to the address below.

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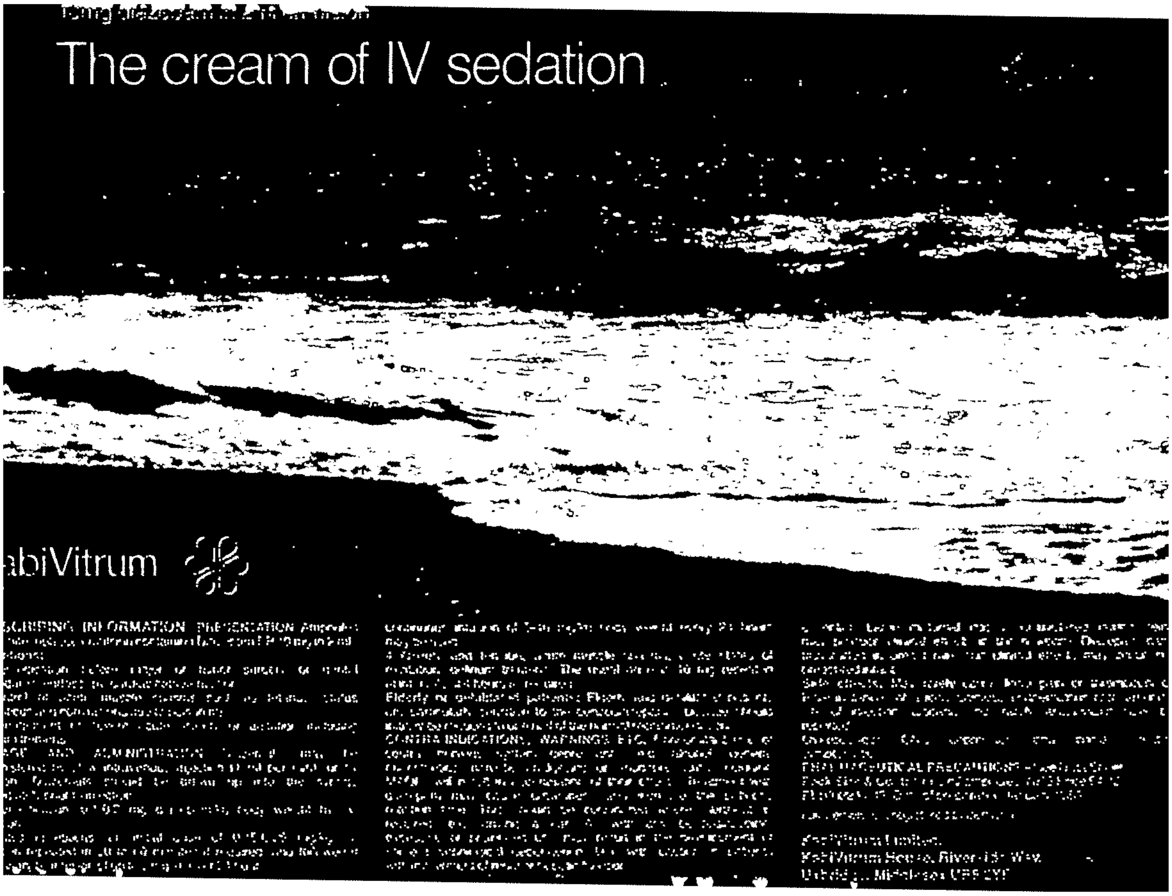
R. Williams



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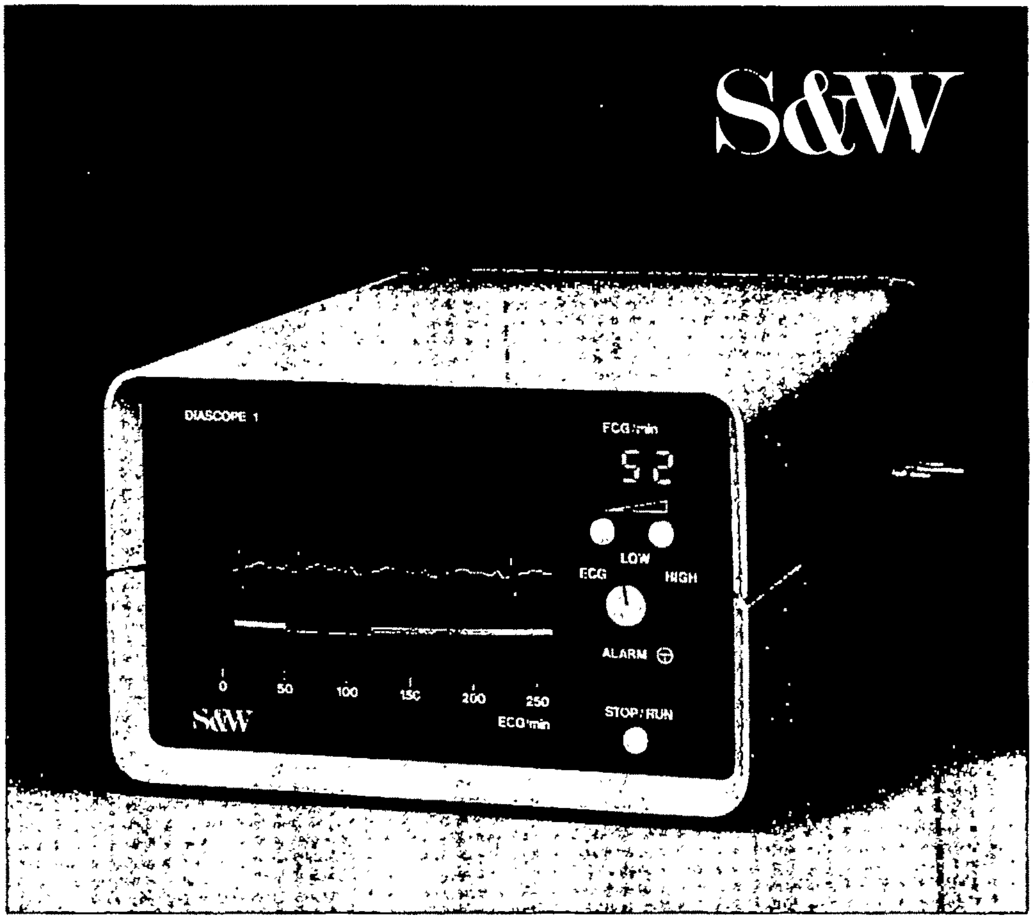

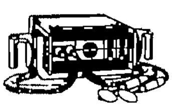
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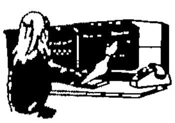
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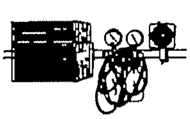
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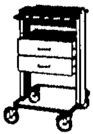
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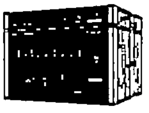
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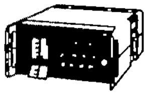
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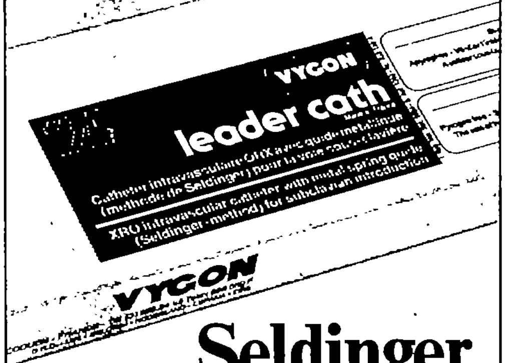

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Pastest Part I FFARCS Practice Exams

By W. Aveling and S. Ingram. Pp. 158. Pastest Service, 1986. £8.50.

There can hardly be a consultant anaesthetist in the land who has not despatched one of his well-read and competent senior house officers to the Part I FFARCS examination certain that the lad will pass with flying colours. When the SHO returns with a sad face, all the consultant can do is scratch his head and wonder what in heaven is going on in Gower Street.

This short, good paperback tells us. The authors, from the Middlesex Hospital and University College Hospital, make the answer clear in their preface. It can be summarised, they say, 'as "organisation and presentation", otherwise referred to as examination technique. The candidate who passed appreciated that the exam is not just about what you have learnt but it is about the ability to recall and relay what you have learnt'.

The book sets out to help candidates present their work to the best advantage in each of the four parts of the examination. The authors begin with a valuable chapter which explains the purpose of the examination, the standard required, the syllabus and, which may come as a surprise to some candidates, the tight 'close marking system'.

The text then deals, in turn, with multiple choice, the written paper and the orals. The chapters have introductions which are excellent value, and they are very well written. The headings give some flavour of the book: Plan of campaign; Listen to the question; Leading the examiner or digging a pit. There are two specimen multiple choice question papers (with answers and explanations), and three specimen written papers with outline answers accompanied by similarly good advice. The clinical oral is covered by 13 case histories. Candidates may often be shown X rays, ECGs, copies of anaesthetic records and actual photographs of patients; these, too, are included, clearly reproduced. Answers and explanations for the case histories are well documented. There is advice on the viva, on further reading and on the choice of reference books, and a short but useful index.

In brief, this book helps both the candidate and the teacher. For your reviewer, testing himself, the experience was humbling, if not actually humiliating. There is more to Part I than meets the eye.

P.V. Scott

Oxygen Free Radicals in Shock

Edited by G.P. Novelli and F. Ursini. Pp. 248. S. Karger, 1985. £62.80.

The role of oxygen free radicals in disease is undoubtedly one of the rapid growth edges of clinical medicine, particularly in relation to critically ill patients. The present volume includes the formal presentations at a workshop on 'Oxygen Free Radicals in Shock' held in Florence during June 1985. The authors and publishers are to be congratulated on the speed and efficiency of their combined endeavours. The result is a very worthwhile, informative and readable product. Unlike many Proceedings, the present volume comes over well in book form and does not require the reader to be expert in the subject.

The authors of the 37 papers come from a wide range of backgrounds which include biochemistry, physiology, pathology, pharmacology, anaesthetics, cardiology and surgery, as well as intensive care. The early chapters are concerned with general aspects of the physiology and biochemistry of oxygen free radicals, the antioxidant enzymes which inactivate them, and the location of their effects on cell membranes. A variety of analytical and physicochemical methods are described, but not in indigestible detail. The main section of the book provides a comprehensive and detailed account of the specific relevance of oxygen free radicals in a variety of experimental shock models. Both *in vivo* and *in vitro* procedures are described. Evidence is advanced for the hypothesis that peroxidative damage to the inner mitochondrial membrane, activated by oxygen free radical intermediates, may characterise circulatory shock. The role of the granulocyte is seen to be central to the production of oxygen free radicals but other, independent, mechanisms also produce vascular endothelial damage. A number of papers deal with the mechanism of action of naturally occurring free radical scavengers, the manner whereby these mechanisms may be overcome during ischaemia and hypoxia and, finally, the evidence that endogenous agents may effectively protect tissues from oxidative damage.

Clearly, the bulk of human studies are reported and the indications are that much of the laboratory information has clinical relevance. As new and less toxic anti-oxidant agents become available, there will be an increase in studies that explore the therapeutic possibilities of this approach.

This volume is well produced and would make valuable, instructive and enjoyable reading for a wide range of doctors and scientists, particularly those involved in anaesthesia and critical care medicine. The only adverse feature is the high cost.

I. McA. Ledingham

Lecture Notes on Anaesthetics. 3rd edition

By John N. Lunn. Pp. 176. Blackwell Scientific Publications, 1986. £8.50.

This book is a member of the popular *Lecture Notes* series, and the fact that there have been three editions and three reprints in seven years testifies to its popularity. It is aimed principally at the undergraduate medical student, although it will also be found to be of value to theatre nurses and ODAs.

The book contains nine chapters and is easy to read and well set out, with clear illustrations. There are a few alterations from previous editions, which take account of the continuous advancement in the specialty of anaesthesia, but these are minor and the overall contents are little changed.

The first eight chapters cover pre-operative assessment and premedication, anaesthetic apparatus, pharmacology with respect to both general and local anaesthesia, applied physiology, coma, monitoring and resuscitation. The ninth is entitled 'A miscellany of special topics', and includes such topics as day case and emergency anaesthesia; it also gives a brief overview as to the scope of the anaesthetist, from cardiac anaesthesia to acupuncture.

Anaesthesia involves a considerable amount of applied physiology and applied pharmacology, and it is pleasing to see these subjects given reasonable coverage. Care of the unconscious patient is included, and it is comforting to see almost 30 pages given over to resuscitation.

The section on pre-operative management is good, but that concerning the postoperative period seems rather brief. It is concerned almost solely with the immediate postoperative period (recovery). My only real criticism is that more should have been included on later postoperative management, with greater detail on postoperative analgesia and fluid balance, both of which are given very little coverage in this edition.

To summarise, the essentials of what the undergraduate needs to know are included in an easy to assimilate fashion. The book is uncluttered by details which are more appropriate to postgraduate fellowship level. I have recommended the previous editions to the undergraduates whom I teach, and I shall continue to recommend this one.

B.J. Pollard

Obstetric Anaesthesia and Analgesia. 3rd edition

By D.D. Moir and J. Thorburn. Pp. 390. Baillière Tindall, 1986. £17.95.

This is the third edition of a book which first appeared in 1976. The format remains unchanged, with 10 chapters, on history, physiology, pharmacology, management of pregnancy and labour (by an obstetrician), systemic and inhalational analgesia, general anaesthesia, regional analgesia, selection of anaesthesia (for specific obstetric circumstances), complications, and resuscitation of the newborn. Also unchanged is the clarity of writing, the ease of reading, the simple line drawings and the extensive list of references at the end of each chapter. But there have been changes, the most notable being that Dr Moir has been joined by his colleague of many years, Dr John Thorburn, as co-author. Also, Dr Martin Whittle has taken over and re-written the chapter on management of pregnancy and labour, which should be read by all anaesthetists.

All chapters have been brought up to date, and some sections rewritten. Particular emphasis is placed on the problems of gastric acidity and failed tracheal intubation. There can be no better description of the problems of general anaesthesia in obstetrics. Regional techniques are extensively reviewed and their benefits stressed.

As the National Board for Nursing, Midwifery and Health Visiting (CMB was so much easier) no longer approves the use of trichloroethylene or methoxyflurane by midwives, the description of these agents and the apparatus for their administration are omitted.

Inevitably the book has increased in size (about 60 pages) and cost, although the latter is modest in present-day terms. The standard of the first two editions has been maintained and the book presents a most readable and up-to-date account of obstetric anaesthesia and analgesia which has been a pleasure to review.

M. MORGAN

Anaesthesia in Obstetrics. Maternal, Fetal and Neonatal Aspects. 2nd edition

By G.A. ALBRIGHT, J.E. FERGUSON II, T.H. JOYCE AND D.K. STEVENSON. Pp. xvii + 622. Butterworths, 1986. £65.00.

The purpose of this book is to '... present a comprehensive, *up-to-date*, readable source of information on all important aspects of obstetric anaesthesia ...'. It is divided into six sections, each of a number of chapters. Section 1 discusses maternal and perinatal mortality, pre-anaesthetic evaluation and record keeping. Section 2 concerns the physiology of pregnancy and labour and the pharmacology of local anaesthetics. The third section deals with analgesia for labour and delivery while the fourth is devoted to major regional anaesthesia, namely, epidural, spinal and caudal. Caesarean section, maternal complications and obstetric problems and their relevance to anaesthesia are discussed in Section 5, while the final Section is devoted to the fetus and neonate.

There have been extensive changes since the first edition. There are now four main authors and ten expert contributors. A novelty has been the inclusion in the text (now two columns per page) of comments by distinguished anaesthetists, an obstetrician and a neonatologist on important and controversial issues, and which do not always agree with the views of the authors. There is a new chapter on anaesthesia for non-delivery surgery during pregnancy and, although this contains discussions on teratogenicity, intra-operative management and postoperative care, is disappointing in that it does not deal with specific problems such as laparotomy during pregnancy, or management of patients undergoing cardiac surgery, including cardiopulmonary bypass. The various sections contain a vast amount of information and deal adequately with most of the problems of obstetric anaesthesia.

However, there is much in this book that British anaesthetists would consider out of context. The inclusion in the chapter on the physiology of labour, of details of pelvimetry, various types of forceps and a whole-page illustration of a vacuum extractor, are quite irrelevant to obstetric anaesthesia. Local anaesthetics are dealt with in some detail; however, is anyone interested in their pharmacokinetics and mode of action, going to turn to a textbook on obstetric anaesthesia? As would be expected from the first author, the cardiotoxicity of bupivacaine is dealt with in detail. While one agrees that this agent is more cardiotoxic than others, there is no discussion as to why maternal deaths associated with the 0.75% concentration were not a problem outside the United States. Similarly, most of the details of the inhalational agents are out of place in such a book. Paracervical block is dealt with in detail, but it has been abandoned in most centres. Much of the chapters on epidural, caudal and spinal anaesthesia could have been omitted without detriment to the subject under discussion. A two-page table on anticonvulsants used in epilepsy is superfluous, although there is much valuable information on the anaesthetic management of the mother with a variety of medical problems, despite the fact that the authors have mistaken alcuronium for atracurium on page 412. Again, one would not expect to find a chapter on fetal growth and development in such a book, while the relevant information in the chapter on fetal monitoring and fetal distress could have been included in that on neonatal resuscitation.

The commonest causes of death in obstetric anaesthetic practice are inhalation of gastric contents and failed tracheal intubation. Not only do these subjects not receive the attention they deserve, but some of the statements made are quite surprising. What on earth is the justification for: 'The application of cricoid pressure (no reference to Sellick) is controversial and is not uniformly practised outside the United States, since it may be ineffective if applied with inadequate pressure ...' (page 353). Are the authors implying that it is only applied with adequate pressure in their country and that everyone else does it ineffectively, if at all? In their discussion of failed intubation (which occupies but one column on page 355), the authors comment that if a tube cannot be passed, then spontaneous breathing is preferable when the airway is difficult. They go on to state that if the airway is easy, an infusion of suxamethonium should be used with 0.5% halothane, similar to endotracheal anaesthesia. Maintenance of paralysis in a patient who cannot be intubated, is highly dangerous and cannot be recommended. Anyway, if the airway is easy, why cannot they be intubated? Neither can one agree with the recommendation that a small dose of a competitive relaxant be given prior to induction and suxamethonium. This will always antagonise the depolarising agent, whatever the dose, will never make intubation easier, but could well make it more difficult.

Have the authors succeeded in their aims? The text is certainly easy to read, interspersed with the right amount of tables and good, clear diagrams. It is also up to date and extensively referenced. But there is much irrelevance and, although the book may find its way into some libraries in the UK, it will not suit individual anaesthetists in this country. This is mainly due to the differences that exist in British and American practice with regard to obstetric anaesthesia, which is also illustrated in another way. The reviewer has recently had the opportunity to read a British text on the subject where, since its previous edition, the number of authors had increased by 100%, the text by 20% and the cost by 44%. The corresponding figures for this present contribution are 400%, 30% and 106%. Well, we always say, things are bigger and better elsewhere!

M. MORGAN

A Colour Atlas of Cardio-Pulmonary Resuscitation Techniques

By G. PAGE, K. MILLS AND R. MORTON. Pp. 96. Wolfe Medical Publications Ltd, 1986. £9.95.

The authors have produced an atlas of cardiopulmonary resuscitation techniques which, when it addresses this subject, is very good. The combination of a stepwise approach to resuscitation, with photographs arranged in sequence, makes points that any amount of text (even combined with line drawings) would find it hard to make.

The presentation of airway control as the first stage of resuscitation is complete and well illustrated, and indicates explicitly how to clear and maintain an airway in an unconscious or semiconscious patient. Similarly, the topics of breathing support and circulation support are well set out, with logical explanations and good photographs to complement the text.

There is also a great deal of additional information in this book which is not logically set out, not well illustrated and not really relevant to cardiopulmonary resuscitation. The description of the basic technique of expired air ventilation wanders off too soon into the specific problems of artificial ventilation of a near-drowning victim in a swimming pool, before the reader has fully grasped the fundamentals of mouth-to-mouth ventilation. The photographs which illustrate the surgical procedures of tracheostomy, chest drain insertion, venous cut-down and central venous catheterisation are meaningless, since there are no illustrations of the relevant anatomy. The pictures of the equipment required for these procedures are too small to be of any real interest. Furthermore, photographs of various pieces of equipment with neither labels nor a functional description, are of no value.

The authors have failed to identify the audience to whom their atlas is directed. For the non-medical first-aider there is a quantity of useful information in the book, although there is much which is irrelevant. For the medical student or junior doctor who is attempting to learn about the subject, there is too little material and what there is, is poorly laid out.

R.D. JACK

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Index of computer programs

004.4.1

Author J.M. Watt (The General Hospital Birmingham, Steelhouse Lane, Birmingham B4 6NH).,

Title ELEC1–4

Description This is a series of four computer tutorials for the teaching of basic electronic theory and the principles behind clinical monitoring equipment. Animated graphics are used to support the text. Progress through the tutorial is contolled by the student.

Language BBC Basic

Computing facility BBC microcomputer.

029.1.24

Author Dr A. Manohin (Institute of Anesthesiology, Medical Centre of Ljubljana, 61105 Ljubljana, Zaloska 7, Yugoslavia). The program was converted to BBC Basic by C.R. Jolleys, Lancaster Royal Grammar School, Lancaster.

Title Distinguishing Upper and Lower Airway Stenosis using PEF/MEF$_{25}$

Description Upper airway stenosis gives rise to respiratory insufficiency. The stenosis should be clearly identified, and distinguished from lower airway disease, as the correct therapy and preoperative preparation of the patient depend on the diagnosis.

Using the quotient PEF/MEF$_{25}$ (peak expiratory flow/maximal expiratory flow at 25% of vital capacity) the stenosis, whether fixed or variable, can be distinguished from lower airway disease whether this is fixed or variable. Following the insertion of the values of PEF and MEF$_{25}$ the ratio is determined and two flow–volume diagrams displayed, normal and actual. Diagnostic comments are then made.

Language BBC and Sinclair Basic

Computing facility BBC microcomputer, disc-based, or Sinclair Spectrum, cassette-based.

030.1.22

Author R.S. Walker (Department of Anaesthesia, Queens Medical Centre, Nottingham).

Title Apache Score Database

Description This program produces a database for the production of Apache scores (Acute Physiologic and Chronic Health Evaluation). When processed, these could permit the prediction of outcome of various pathological conditions and could also generate prognostic indices.

The program is menu-driven and allows data to be stored in user-named files. Existing files can be extended and edited, which allows the insertion, alteration and deletion of individual patient records. A printer facility is also included.

Language BBC Basic

Computing facility The present program runs on a BBC Model B via a disc drive using an Acorn DFS, but can be easily amended for the ADFS.

Details of programs to be included in this catalogue should be sent to Dr M.J. Harrison, Department of Anaesthesia, Nottingham City Hospital, Hucknall Road, Nottingham NG5 1PB. They should be of relevance to anaesthesia or intensive care. Programs that involve the application of the basic sciences, or teaching, will also be accepted. Further details of the programs can be obtained from the department or person directly involved.

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The collator of this section is Dr L. Kaufman, MD, FFARCS, Anaesthetic Office, The Rayne Institute, University College, University Street, London WC1E 6JJ. Dr Kaufman is prepared to provide on request, and at a modest charge, a new additional service to our readers. References from January 1984 have been entered on data base. The data are held on Dbase II, and are available on disk, together with a program providing search facilities. Enquiries direct to Dr Kaufman at the above address please.

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Physiology

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Physiology

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Physiology

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Treatment and medication

Neuroendocrine tumours of the gut: long-term therapy with the somatostatin analogue SMS 201–995. ANDERSON, J.V., BLOOM, S.R. *Scandinavian Journal of Gastroenterology* 1986; **21:** 115.

Different effects of metoclopramide and domperidone on arginine-vasopressin secretion in man. CHIODERA, P., VOLPI, R. *et al. British Journal of Clinical Pharmacology* 1986; **22:** 479.

Acute renal failure. 2. Management of suspected and established disease. FILDES, R.D., SPRINGATE, J.E., FELD, L.G. *Journal of Pediatrics* 1986; **109:** 567.

Rapid replacement of serum albumin in patients receiving total parenteral nutrition. HARDIN, T.C., PAGE, C.P. *Surgery, Gynecology and Obstetrics* 1986; **163:** 359.

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Pain

Physiology

The many possible roles of opioids and related peptides in stress-induced analgesia. AKIL, H., YOUNG, E. *et al. Stress-Induced Analgesia.* Ed. D.D. KELLY. New York Academy of Sciences, 1986; p. 140.

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Relation of stress-induced analgesia to stimulation-produced analgesia. TERMAN, G.W., LIEBESKIND, J.C. *Stress-Induced Analgesia.* Ed. D.D. KELLY. New York Academy of Sciences, 1986; p. 300.

Treatment and medication

Comparison of the analgesic effect of a corticosteroid and paracetamol in patients with pain after oral surgery. OLSTAD, O.A., SKJELBRED, P. *British Journal of Clinical Pharmacology* 1986; **22:** 437.

Other

Treatment and medication

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Obituaries

Dunnill, Dennis Estyn, MB, BS, MRCS, LRCP, DA, formerly Consultant Anaesthetist at Guy's Hospital. Qualified from the University of London in 1929.

Hammerton-Fraser, Allan Miller, MB, BChir, LMSSA, DA, formerly Head of Clinical Measurements, RAF Institute of Aviation Medicine, Farnborough. Qualified from the University of Cambridge 1954.

Gergis, Luther Abdelmalek, MB, BCh, FFARCSI, formerly Consultant Anaesthetist at the Royal National Orthopaedic Hospital, Stanmore. Qualified from the University of Cairo.

Moran, John Leo Christopher, MB, BCh, BAO, FFARCSI, FFARCS, formerly Consultant Anaesthetist at Altnagelvin Hospital, Derry. Qualified from University of Belfast in 1973.

Todd, Christopher Granville, MB, BCh. Qualified from the University of Belfast 1982.

International congress calendar

1987

2–4 April. Tokyo. *34th Annual Meeting, Japan Society of Anaesthesiologists.*
Information: Dr Kenjito Mori, 5F, TY Building, 18–11 Hongo 3-chome, Bunkyo-ku, Tokyo.

7–9 May. Paris. *VIth Annual Meeting of the European Society of Regional Anaesthesia.*
Information: MAPAR, Departement d'Anesthesiologie, Hopital de Bicetre, 94270 Kremlin-Bicetre, France.

11–14 May. Bucharest. *International Symposium of Anaesthesia and Intensive Care.*
Information: Romanian Society for Anaesthesia and Intensive Care, 10 Progresului Strasse, 70754 Bucharest, Romania.

20–23 May. Halifax, Nova Scotia. *Society for Obstetric Anesthesia and Perinatology,* 19th Annual Meeting.
Information: Dr D. Writer, SOAP 1987, Grace Maternity Hospital, 5821 University Avenue, Halifax, Nova Scotia, Canada, B3H 1B3.

19–21 May. Gottingen, W. Germany. *European Association of Cardiothoracic Anaesthetists.*
Information: Professor D. Kettler, Georg August Universitat, Robert Koch Strasse 40, D-3400, West Germany.

28–31 May. Chania, Crete. *7th Congress of the Greek Society of Anaesthesiologists.*
Information: Greek Society of Anaesthesiologists, 34 Dragoumi Street, 115 28 Athens, Greece.

31 May–8 June. Paris, France. *International Seminar on Advances in Anesthesiology.*
Information: George Silvay, MD, PhD, Department of Anesthesiology, Mount Sinai Medical Center, 1 Gustave L. Levy Place, New York 10029, USA. Tel. (212) 650-5954.

7–11 June. Meridien Hotel, Montreal, Canada. *First International Shock Congress and Tenth Annual Conference on Shock.*
Information: Shock Society, Dr Sherwood M. Reichard, Medical College of Georgia, Augusta, GA 30912, USA.

13–18 June. Calgary, Alberta. *Canadian Anaesthetists' Society Annual Meeting.*
Information: Mr Lloyd H. Mayeda, 94 Cumberland Street, Suite 901, Toronto, Ontario, Canada M5R 1AS.

14–17 June. Viscount Hotel, San Diego, California. *Computers in Critical Care and Pulmonary Medicine.*
Information: Richard M. Peters, MD, Department of Surgery, UCSC Medical Center, 225 Dickinson St, San Diego, California, USA 92103–9981.

17–19 June. Nijmegen, Holland. *International Congress in Honour of Professor Dr. J.F. Crul.*
Information: Miss I. Baltussen, Department of Anesthesiology, University of Nijmegen, St. Radboudziekenhuis, Geert Grooteplein zuid 10, P.O. Box 9101, 6500 HB Nigmegen, The Netherlands.

20–25 June. Montreal, Canada. *XIIIth International Symposium on Cerebral Blood Flow and Metabolism.*
Information: Sorelcomm (1985) Inc., Conference Secretariat, 1425 Dorchester Boulevard West, Montreal, Canada H3G 1T2.

29 June–3 July. Linköping. *19th Scandinavian Congress.*
Information: Department of Anaesthesiology, University Hospital, S-581 Linköping, Sweden.

20–23 July. London. *Second International Symposium on the History of Anaesthesia.*
Information: Secretariat of the Second International Symposium on the History of Anaesthesia, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA, UK.

2–7 August. Hamburg. *Vth World Congress on Pain.*
Information: Hamburg Messe & Congress, Postfach 30 24 80, 2000 Hamburg 36, Federal Republic of Germany.

4–8 August. Quality Inn, Nelson. *Conference of Anaesthetists of New Zealand Annual Meeting.*
Information: Dr M.L. Farrant, Secretary CANZ 87, Nelson Hospital, Private Bag, Nelson, New Zealand.

23–28 August. Sydney, Australia. *10th International Congress of Pharmacology.*
Information: The Secretariat, 10th IUPHAR Congress, G.P.O. Box 2609, Sydney, N.S.W. 2001, Australia.

26–28 August. Edinburgh. *Edinburgh Anaesthesia Festival.*
Information: The Department of Anaesthetics, Royal Infirmary, Edinburgh EH3 9YW, UK.

27–30 August. Kobe, Japan. *International Symposium on Cardiovascular Anesthesiology and 8th Annual Meeting of Japan Society of Circulation Control in Medicine.*
Information: Secretariat, ISCVA/SCC, Japan Convention Services Inc., Kansai Branch, Sumitomo Seimei Midosujui Building, 1 DF 4-14-3, Nishitemma, Kita-ku, Osaka 530, Japan.

2–5 September. Singapore. *Fifth Association of South East Asian Nations Congress of Anaesthesiologists.*
Information: Congress Secretariat, 5th ASEAN Congress of Anaesthesiologists, World Express PTE Ltd, 114 Middle Road, 05-01 Singapore 0718.

3–6 September. Ghent, Belgium. *European Academy of Anaesthesiology 9th Annual Meeting.*
Information: Professor Dr G. Rolly, Department of Anaesthesiology, Academic Hospital, De Pintelaan 185, B-9000 Ghent, Belgium.

10–11 September. Sheffield. *Annual Scientific Meeting, Association of Anaesthetists of Great Britain and Ireland.*
Information: The Honorary Secretary, The Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA, UK.

15–18 September. Paris. *IIIrd International Symposium on Pediatric, Surgical and Neonatal Intensive Care.*
Information: S.O.C.F.I., 14 Rue Mandar, 75002 Paris, France.

10–14 October. Atlanta. *Annual Meeting, American Society of Anesthesiologists.*
Information: John Andes, Esq., 515 Busse Highway, Park Ridge, IL 60068, USA.

22–24 October. Posnan, Poland. *Symposium—DYDAKTYKA '87.*
Information: The Organizing Committee of Symposium DYDAKTYKA '87, Institute of Anaesthesiology and Intensive Therapy, ul. Przybyszewskiego 49, 60-355 Poznan, Poland.

22–24 October. Berlin. *2nd International Steglitz Symposium.*
Information: Priv.-Doz. Dr K. Reinhart, Klinikum Steglitz, Freie Universitat Berlin, Hindenburgdamm 30, 1000 Berlin 45, Federal Republic of Germany.

16–21 November. Rotterdam, The Netherlands. *The 4th International Symposium on Applied Physiology in Cardio-Respiratory Emergencies.*
Information: Omar Prakash, MD, Erasmus University, Rotterdam, P.O. Box 1738, 3000 DR Rotterdam, The Netherlands.

13–17 December. New York Hilton Hotel, New York. *40th Postgraduate Assembly in Anesthesiology.*
Information: Mr Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 30 East 42nd Street, Suite 1501, New York, NY10017, USA.

1988

11–13 May. Sydney, Australia. *5th International Dental Congress on Modern Pain Control.*
Information: Australian Convention and Travel Services (ACTS), G.P.O. Box 1929, Canberra, A.C.T. 2601, Australia.

16–20 May. US Grant Hotel, San Diego, California. *Computing in Anesthesia and Intensive Care.*
Information: Office of Continuing Medical Education, M-017, UC San Diego School of Medicine, La Jolla, CA 92093, USA. Tel. (619) 534-3940.

22–28 May. Washington, DC. *9th World Congress of Anesthesiology.*
Information: American Society of Anesthesiologists, 515 Busse Highway, Park Ridge, IL 60068, USA.

15–16 September. Southampton. *Annual Scientific Meeting, Association of Anaesthetists of Great Britain and Ireland.*
Information: Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA, UK.

1 October. Pavia. *National Congress, Societa Italiana di Anest, Rianimazione e Terapia Intensiva.*
Information: Dr G. Conti, SIARRTI, Universita degli Studi, La Sapienza di Roma, Viale del Policlinico, 00161 Roma, Italy.

18–21 October. Ballarat, Victoria. *Annual General Meeting, Australian Society of Anaesthetists.*
Information: The Secretariat, Australian Society of Anaesthetists, P.O. Box 345, Paddington, N.S.W. 2021, Australia.

1989

3–8 September. Kyoto, Japan. *Fifth World Congress on Intensive and Critical Care Medicine.*
Information: Dr Keisuke Amaha, Fifth World Congress on Intensive and Critical Care Medicine, c/o Japan Convention Services Inc., Nippon Press Centre Building, 2-2-1 Uchisaiwai-Cho, Chiyodu/Ku, Tokyo 100, Japan.

1990

9–15 September. Warsaw. *VIIIth European Congress of Anaesthesiologists.*
Information: The Organising Committee, VIIIth European Congress of Anaesthesiologists, c/o Polish Society of Anaesthesiology & Intensive Therapy, ul. Kasprzaka 17a, 01-211 Warsaw, Poland.

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The Annual Conference of Linkmen of the Association of Anaesthetists of Great Britain and Ireland, 1986

The Annual Conference of Linkmen was held in the City University, London, on Wednesday 17 September 1986.

The President, Dr T.B. Boulton, was in the chair. He welcomed Professor M.D. Vickers, Immediate Past President, and the 163 Linkmen present. Dr Boulton reminded Linkmen that the Association had been the leading influence in the advancement of anaesthesia as a specialty since the 1930s. He stressed the importance of the Linkmen organisation and of the value placed by Council on the information and opinions received from Linkmen throughout the year and at the Annual Conference.

9 Bedford Square

Dr Boulton reported that the move of the Association headquarters from Tavistock House to 9 Bedford Square had taken place in November 1985. He described and showed illustrations of the accommodation and facilities provided in this listed Georgian house. Dr Boulton reminded Linkmen that the lease for, and the refurbishment of, the house had been financed from Association funds. He went on to inform Linkmen of the generous donations made to the Appeal Fund by specialist, regional and local societies of anaesthesia, by members and by industry. The building has now been completely refurbished and is functioning as the Association's administrative headquarters. The furnishing programme has commenced and will be completed during the next few months. The Members' room, library and museum will be functional in the near future and the centre will become fully operational following further development of the educational programme of seminars and small discussion groups. There will be an official opening of the house in the spring of 1987.

Dr W.R. MacRae, Vice-President, outlined the many responsibilities and necessary domestic consequences associated with the setting up and daily management of a property such as 9 Bedford Square. The increased administrative workload placed on Council and the executive officers was acknowledged and Dr MacRae particularly thanked the President, Dr T.B. Boulton, and the Honorary Secretary, Dr M.T. Inman, who have borne the brunt of this additional work. Dr MacRae considered that property was the best investment for Association funds and he acknowledged the guidance and advice given by Dr W.D. Wylie, Dr P.J. Helliwell, Past Presidents, and Mr E. Warburton, Association Financial Adviser, in the acquisition of 9 Bedford Square.

Dr MacRae then presented details of the financial state of the Appeal Fund and stated that the target of £600,000 had been achieved. The tremendous contribution made by Professor Rosen, Vice-Chairman of the Appeal, in raising funds from members, industry and charitable organisations, was acknowledged.

Abbott Laboratories had made the first donation, followed by major contributions from Draeger, the Wolfson Foundation, the Wellcome Foundation, Portex, British Oxygen Company, Janssen Pharmaceuticals Ltd and Imperial Chemical Industries. The imminent closure of the appeal was announced and a final appeal made to those who had not yet donated. The names of donors are to be recorded in a commemorative *Book of Founder Donors*, and Linkmen were encouraged to subscribe to ensure recognition.

Donors who had not received an acknowledgment letter from Dr MacRae by the end of 1986 were requested to contact the Association office to ensure that their names were being recorded in the Founder Donors Book. The President thanked Professor Rosen for his magnificent achievement in reaching the Appeal's target.

Smoking and anaesthesia

Dr R.M. Jones (*Guy's Hospital*) informed Linkmen of the Association's initiative in setting up a joint working party with the Health Education Council to mount a campaign to reduce self-inflicted morbidity from smoking in patients undergoing anaesthesia and surgery. He thanked Professor Rosen, President Elect, for his guidance on this project. Dr Jones said that about one-third of adult patients who present for surgery are cigarette smokers. The evidence is overwhelming that this habit is associated with an increased incidence of intra and postoperative complications. There is also now substantial evidence to support the fact that a period of abstinence from smoking prior to surgery, can decrease the risk of peri-operative morbidity and mortality in smokers. A period of abstinence for as little as 12 hours will have beneficial effects, but a minimum period of 6 weeks is required to make a significant impact on the incidence of postoperative respiratory complications. This message must be given to patients. Advice to stop smoking, given in hospital and reinforced in follow-up clinics, has been shown to be effective in persuading patients to stop smoking. Considered, sensible advice by anaesthetists and surgeons could help towards this end.

Dr Jones asked Linkmen for their views on the two draft advisory leaflets, the one directed at health care professionals and the other directed at patients, which had been drawn up by the joint working party. Initially these leaflets would be circulated in a few districts as a pilot scheme to test the efficacy of this approach.

Discussion. Several Linkmen, including Dr M.R. Bryson (*Newcastle*), Dr D.P. Cartwright (*Derby*), Dr J. Hurdley (*South Birmingham*) and Dr D.C. Hughes (*Chertsey*), participated in the discussion. There was general agreement with the advice outlined in the leaflets. However, anxieties were expressed that it might be inappropriate to exert psychological pressure on patients to stop smoking in the immediate preoperative period. The important role of surgeons was stressed, particularly the opportunity for them to offer advice to patients at an earlier stage than was possible by anaesthetists. The President stated that the Association's pamphlet, *Your anaesthetic*, advised patients to stop smoking. Dr A. Padfield (*Sheffield*) considered that nurses' attitudes to smoking were relevant and that they also should encourage patients to give up the habit. Professor Rosen emphasized that it is the duty of anaesthetists to improve patients' fitness for anaesthesia and surgery and to inform them how risks could be reduced. He then asked Linkmen for volunteers to take part in the pilot scheme. The discussion closed with conference giving approval to Council's course of action.

Domiciliary visits

Dr E.B. Lewis (*Kent*) stated that the definition of a domiciliary visit was laid down in the National Health Service document on the terms and conditions of service for hospital, medical and dental staff (England and Wales), paragraph 140. The maximum number of domiciliary visits qualifying for payment, was 300 *per annum*. Some anaesthetists perform as many as 5 or 6 domiciliary visits on average *per annum*, usually for pain-relief work. He stressed that a domiciliary visit must be initiated by a General Practitioner who must sign the appropriate form to ensure payment of the fee, currently £28.25, plus a fee for use of apparatus. When a Consultant supplies drugs from his personal stock he should replenish these through the hospital pharmacy. A record should be kept of visits and treatment undertaken. Domiciliary visits for pre-operative anaesthetic assessment were not recognised by the DHSS. Difficulties have been experienced in obtaining payment for a course that requires several anaesthetics, such as for electroconvulsive therapy.

Discussion. Dr J.R. Jenkins (*Great Yarmouth*) asked Dr Lewis if a visit to a General Practitioner Hospital qualified as a domiciliary visit. A similar question with respect to a hospice was put by Dr P.F. Copeland (*Newcastle*). In reply, Dr Lewis said that a visit to other NHS hospitals did not qualify, while one to a private institution did. If regular visits were made to NHS hospices, additional fractions of a notional half day (NHD) should be negotiated for this work, rather than an attempt made to use the domiciliary visit scheme. Dr J.S. Mason (*Mansfield*) considered that it was more efficient to see patients at home for pre-anaesthetic assessment. Dr Lewis replied that the DHSS did not regard domiciliary visits as an efficient use of finances and resources, and that difficulties were being experienced in maintaining finances for the present level of domiciliary service. This view was supported by Dr D.W. Wood (*Durham*).

Other contractual matters

Dr E.B. Lewis explained the limitations of the NHS superannuation scheme in providing personal accident insurance cover for doctors who performed flying-squad duties. Benefits in the event of death or severe injury while performing these duties, were based on actual pay at the time of injury and not on expected earnings. Young doctors and their families, particularly those with less than 5 years' service in the NHS, were at risk of inadequate financial compensation. He advised that private personal accident cover should be taken out by all doctors and that suitable schemes were offered by the British Medical Association Insurance Service and the Medical Insurance Agency. Any benefit paid under these schemes would have no effect on NHS entitlement, provided the premiums had been paid from private funds or departmental free monies. This was most important, as benefits were paid to the policy-holder, who was responsible for the premium, and this must not be an NHS agency. Dr Lewis confirmed that this lack of insurance cover also applied to doctors who participate in inter-hospital transfer of patients. Dr Lewis then commented on the increase in subscriptions required by medical defence organisations. He said that these increases had probably been taken into account by the Review Body when the latest pay award had been made. He also outlined the potential disadvantages to doctors, of reimbursement of these dues by Health Authorities.

National Anaesthetic Adverse Reaction Advisory Service (NAARAS)

Professor W.S. Nimmo detailed the constitution of the advisory committee of this Service and stated that its aims were to investigate and collect data on the immediate hypersensitivity type of adverse reactions to anaesthetic drugs and other intravenous therapy. The reporting clinician was given a full report which included the probable cause of the reaction, and was advised on future anaesthetic management. Future aims were to build up a data bank, determine the effects of specific therapy and improve the specificity of advice offered.

Discussion. Dr J. Watkins (*Sheffield*), NAARAS Coordinator, said that there were problems to evaluating adverse reactions, particularly when they were associated with polypharmacy. He requested more detailed reports from clinicians. In reply to questions from Dr H.L. Goldwater (*Manchester*) and Dr M.J. Wade (*Bradford*), Dr Watkins gave advice on which reactions should be reported and confirmed that an annual report of NAARAS activities was available.

Adverse drug reaction reporting

Dr J. Lumley (*London*), a member of the Joint Faculty Association and Committee on Safety of Medicines (CSM) Adverse Drug Reaction Reporting Working Party, stated that the CSM has been considering ways of improving the reporting of adverse drug reactions. Many anaesthetists regard the yellow card system as unsuitable and Dr Lumley informed the conference that a new red card had been specifically designed for reporting acute reactions such as occur in anaesthesia, in accident and emergency departments and in intensive care units. A pilot study of the use of the new card is to be undertaken by the CSM in two regions, and Dr Lumley requested assistance from Linkmen in this evaluation. She also indicated that there had recently been an increase in the number of reports received by the CSM and it was hoped that there would be, in future, an improvement in the adverse drug reaction reports.

Discussion. Dr Mann (*CSM*), commenting on the value of the liaison with anaesthetists, requested Linkmen to inform him of problems experienced with the card system of reporting adverse reactions. Recognition of the different time scale of adverse reactions that occur in anaesthesia, as compared to those that occur in other specialties, was welcomed by Dr D.P. Cartwright (*Derby*). The President considered that discussion of this subject at the 1985 Conference had promoted improvement in the reporting system, and that this illustrated the value of the Linkman Conference.

The work of the Faculty

Mr S.N. Alan, Secretary of the Faculty of Anaesthetists, Royal College of Surgeons of England, stated the statutory requirements for this body. The criteria for General Professional Training (GPT) recognition were reviewed. The essential anaesthetic experience and facilities which should exist within a district to allow Schedule I designation for GPT training, were outlined. He explained that Schedule II recognition was granted when the range of anaesthetic experience available was limited. This did not, however, imply that the service was second class, and emphasized that secondment to obtain extra experience was acceptable. Although a Faculty visit, usually by members of the Board of Faculty, may result in a threat to withdraw recognition for GPT purposes, this was not intended to be inquisitorial but to confirm a balance between training and service commitments. Such visits had proved a useful lever in improving sub-standard facilities within a district.

Higher Professional Training (HPT) was supervised by the Joint Committee on Higher Training of Anaesthetists (JCHTA). Inspections similar to those for GPT, were carried out by Faculty visitors to confirm the appropriateness of individual HPT schemes. Emphasis was placed on District General Hospital (DGH) experience. The Faculty also assessed Senior Registrar suitability for enrolment for HPT and accreditation.

An invitation was extended by Mr Alan to Linkmen to attend the FFARCS examination as observers.

Mr Alan concluded by describing the Faculty's role in manpower discussions, on advisory appointment committees and the Faculty involvement on national bodies.

Discussion. The President remarked that an excellent hospital with a poor administration may receive an adverse report and confirmed that Schedule II recognition was an expression of the experience offered. In reply to a question from Dr M.T. Inman (*Honorary Secretary*) on recognition towards HPT of experience gained in Third World countries, Mr Alan said that this could be considered and depended on the training potential. The President stressed that the possession of HPT accreditation was not essential to attain a Consultant appointment, and that a common-sense approach was adopted by advisory appointment committees and the Faculty. Dr C.D.D. Hutter (*Derby*) requested an explanation for the large increase in Faculty fees. Mr Alan detailed the reasons for this increase and referred Dr Hutter to the *Dean's Newsletter*, which stressed the necessity for investment in the future. He went on to say that the period of reduced fees for trainees will, in the future, be increased from 3 to 5 years.

The President emphasized that subscriptions were now received directly by the Faculty of Anaesthetists, rather than through the Royal College of Surgeons.

Equipment procurement and maintenance

Dr P.L. Jones (*Cardiff*) stated that advice from the DHSS on equipment procurement and management was contained in the *Health Equipment Information Document, HEI No. 98, Management of Equipment.* He reviewed the general principles of good management of equipment and considered that an accurate inventory should be taken of all existing apparatus within a district. This would enable identification of deficiencies, of replacement costs and would also aid the drawing up of a statement of predicted future requirements. He stressed the importance of the selection of equipment by users, who should take into account its predicted life, installation costs and the revenue consequences of servicing, and necessary consumables. The proposed purchase of equipment should be justified by a statement of need, using form MEQ1, and acceptance procedures should be standardised.

Maintenance of equipment should be carried out either by manufacturers or by in-house technicians. These staff should be trained by manufacturers and have access to official service manuals and replacement parts, as recommended by the British Anaesthetic and Respiratory Equipment Manufacturers Association (BAREMA). An after-service checklist should be provided on individual items of equipment, to enable the first user to ensure correct function. There should also be a regular maintenance schedule within a district.

The nomination of a clinician as departmental equipment controller and the provision for the employment of a superviser of technical servicing were recommended. Dr Jones concluded by stressing the need for an agreed policy to enable unserviceable and unusable equipment to be replaced.

Mr C. Bray (*DHSS*) described the structure of the DHSS procurement directorate, Director, Mr T. Critchley, and the roles of the Scientific and Technical, and Commercial branches. The directorate's objectives were to obtain the best value for money. The Scientific and Technical branch carries out evaluation and comparison tests, gives explanations on the use of equipment and makes comments on standards. To date, many technical devices used by anaesthetists, including lung ventilators, cardiac defibrillators and monitors, have been evaluated and the results published in the HEI documents. Mr Bray requested Linkmen's views on the value of the equipment evaluation scheme.

A manufacturers' registration scheme, regularly updated, had been introduced by the DHSS. Health Authorities were encouraged to select a manufacturer from this register. Mr Bray then explained the Medical Equipment Questionnaire (MEQ) system and drew Linkmen's attention to the documents which had been prepared by the DHSS on quality assurance and on choosing medical equipment.

Discussion. Dr I. McLellan (*Leicester*) commented on the difficulties of obtaining the data base for a departmental inventory and emphasized the difficulties of in-house servicing. Dr H.L. Goldwater (*Manchester*) expressed concern that equipment could be purchased when it did not satisfy established standards. Dr M.R. Bryson (*Newcastle*) remarked that equipment had not always been evaluated when it was demonstrated at exhibitions. Dr P.L. Jones (*Cardiff*) gave some further guidance on in-house maintenance schemes. Professor M.D. Vickers (*Cardiff*) said that all equipment in public hospitals could be, and should be, officially approved, as is the practice in France.

Manpower

Dr M.M. Burrows (*Honorary Treasurer*) stated that the consultative document *Hospital Medical Staffing – Achieving a Balance*, initiated by Mr Barney Hayhoe, Minister of State for Health, had been published in July. The document had been put forward as an achievable solution to the long-standing manpower problem. Dr Burrows said that this problem was not as severe in anaesthesia as in other specialties, such as general medicine and surgery. The intention was that a young doctor would have settled on a career option by 28 or 29 years of age. The proposed scheme allowed for an increased period in the SHO grade to obtain a higher qualification. It was also proposed that early formal regular careers counselling would be given at this stage. Progression to the limited number of Regional Registrar posts would be reserved for United Kingdom graduates eligible to seek a Consultant career in this country. All other Registrar posts would be designated District Registrar posts and, as these become vacant, they would be reserved for overseas graduates in training. Dr Burrows emphasized that District Registrar posts would provide equal training opportunities and participation in rotations to Regional posts.

A new Intermediate career grade would be introduced to provide a service need. The Intermediate grade post would have security of tenure and an entitlement to continuing education. The size of this grade would not exceed 10% of the numbers of Consultants. There would be a 'safety net' for those Consultants with small numbers of supporting staff.

Recommendations on Senior Registrar numbers had not been included in the remit of the joint committee and would be considered by the Joint Planning Advisory Committee (JPAC).

There would be a total of 100 new Consultant posts equally divided between general medicine and general/traumatic and orthopaedic surgery. There would be no additional Consultant posts in anaesthesia. However, the current average 2% increase in Consultant numbers would be maintained. Early or partial retirement for Consultants had been proposed. Dr Burrows said that the opportunity presented by this document to influence the manpower situation had been accepted by the CCHMS, but solutions to all the problems had not yet been found.

Discussion. Dr R. Bose (*Neath*) said that there was already a lack of applicants for SHO posts in anaesthesia, and considered that this was caused by the attraction of general practice vocational training schemes. Dr Burrows replied that pre-registration housemen were perhaps being influenced by disillusioned Registrars in medicine and surgery. Dr D.C. Hughes (*Chertsey*) supported the Intermediate grade, which he felt could promote a balance between training and service needs and provide a role for those who do not aspire to Consultant status. Dr P.E.G. Tannett (*Wakefield*) made the comment that there were not enough posts in anaesthesia. Dr S.D. Dalal (*London*) considered that the FFARCS should be a requirement for the appointment to the Intermediate grade. Dr R.W. Griffin (*Colchester*) asked whether this grade could be held on a part-time basis and received confirmation on this point from Dr Burrows. Dr C.K. Adam (*East Fife*) enquired whether the Intermediate grade was in addition to, or a replacement for, SHOs and Registrars. Dr Burrows replied that the new post was not supernumerary and that there would be a moderate increase in SHO and a reduction in Registrar posts. Dr J.M. Ward McQuaid (*Grimsby*) said that rotational posts would help to prevent shortage of SHOs. Dr C.E. Blogg (*Oxford*) expressed the opinion that there were too many medical graduates.

Honorary Secretary's update

Dr M.T. Inman (*Honorary Secretary*) thanked Linkmen for their correspondence throughout the year, which had generated topics for discussion at the Conference. He asked Linkmen to inform the Association of dates of other anaesthetic meetings and he informed Linkmen of a meeting on 'Safety of Anaesthetic Equipment' to be held by the Institute of Mechanical Engineers.

The Private Practice Committee continues to press for separation of benefits paid by the Provident Associations and for direct payment by these organisations to Consultants. Over half the Linkmen present indicated that there were private group anaesthetic practices in their district.

Concern had been expressed by the Junior Anaesthetists' Linkmen about the effects of fatigue on anaesthetists' clinical performance and on patient safety. Dr Inman stated that it was the trainee's responsibility to report his clinical incapacity due to fatigue and other reasons, to a Consultant, who would make the necessary managerial decisions.

Many letters had been received from members requesting the Association to support the Operating Department Assistant (ODA) grade. Two particular issues which had been raised, related to the handling of controlled drugs, and terms and conditions of service of the ODA. These problems were appreciated by Council, who have agreed to lend their support to ODAs.

The Honorary Secretary introduced the charter for children in hospital which had been drawn up by the National Association for the Welfare of Children in Hospital, and said that Association approval for the charter was being sought. Council had requested the views of Linkmen to help them in its deliberations. Several Linkmen, including Dr A.B. Lodge (*Northampton*), expressed reservations concerning approval, particularly in respect to the interpretation of the statement 'children in hospital shall have the right to have their parents with them at all times provided this is in the best interest of the child'. The President requested Linkmen to inform the Association of their opinions in writing. The matter was left to Council, who will take into account the views expressed and will come to a decision.

The Honorary Secretary exhorted Linkmen to continue to write to the Association on any matter which worried them.

Open forum

Short-style anaesthetics at Milton Keynes

Dr M.J. Cowen (*Milton Keynes*) described the work pattern of the 10 Consultants at Milton Keynes. The Junior staff provision necessitated a major on-call commitment at night by Consultants. Consultants have seven day-time sessions, a day off after being on call, and a rotating work programme. He explained the advantages and disadvantages of this system to anaesthetists.

Dr J.H. Wright (*Tameside*) and Dr J.H. Breed (*Manchester*) participated in the discussion. The main points raised concerned cover for Consultant absences and the length of time a Consultant would be satisfied, or able, to work in this manner.

Difficulties in recruitment of ODAs

Dr E.A. Maher (*Suffolk*) supported the case for improving the terms and conditions of service for ODAs. Dr E.B. Lewis (*Kent*) added his support and said that the Central Committee for Hospital Medical Services was also concerned.

Rationalisation of surgical services

Dr A.M. Rollin (*Epsom*) outlined a policy which was being imposed on her district, in which the movement of ENT and eye surgery from one district to another was creating implications for training and service. Dr P.F. Copeland (*Newcastle*) said that this was also occurring in Newcastle, and that rotational schemes were the answer to training deficiencies.

BUPA benefits and bad debts
This question was raised by Dr M. Bryson (*Newcastle*) and Dr R.G. Snow (*Chester*). The Association's policy to press for direct payment of the Consultant by the Provident Associations, was reiterated.

Informed consent
Dr J.L. Jenkinson (*Edinburgh*) questioned the necessity to obtain informed consent from patients before procedures such as local anaesthetic procedures.

Interview visiting by Registrars and Senior Registrars
Dr M. Marshall (*Newcastle*) made comments on the practice of trainees visiting hospitals before they had been shortlisted for an appointment.

BBC Save a Life Campaign
Dr P. Burridge (*Rochdale*) gave his views of the value of training the general public in resuscitation techniques, and informed Linkmen of the BBC television series on this topic.

Anaesthetic charts and records
Dr A. Padfield (*Sheffield*) asked for advice on how anaesthetic records could be retained in the patient's case notes.

The Meeting was declared closed by the President, Dr T.B. Boulton, who thanked Linkmen for their valuable and informed comments.

Anaesthesia, 1987, Volume 42, page 465

Errata

Anaesthesia, 1986, Volume 41, page 1154

Anaesthesia for extracorporeal shock wave lithotripsy

In this letter from Drs R.F. Seed, H. Khalil and R. Prabhu, the inequalities in the first sentence of paragraph two (lines 17 and 18 of the letter) were reversed. The correct version should read:

> In a series of 100 patients randomly allocated into two equal groups and ventilated with N_2O and O_2 (33%) either by intermittent positive pressure ventilation (IPPV) or by high frequency jet ventilation (HFJV) we found, in the latter group, that blood gases were unsatisfactory ($Pa\text{CO}_2 > 6.3$ kPa and $Pa\text{O}_2 < 11.2$ kPa) in 17 patients, mainly in the heavier males.

Anaesthesia, 1986, Volume 41, page 1286

Courses in Anaesthesia: FFARCS Part 3

The month for the revision course 'Clinical Anaesthesia' was given incorrectly as August instead of May. The correct entry should read:

LEICESTER Postgraduate Medical Centre, Leicester Royal Infirmary, Leicester, LE1 5WW.	11–15 May 1987 1 week, whole time, revision course 'Clinical Anaesthesia'	£35 NO CHARGE FOR DOCTORS WITHIN TRENT REGION	The Secretary, Department of Anaesthesia, Leicester Royal Infirmary, Leicester, LE1 5WW

Anaesthesia, 1986, Volume 41, page 1257

Absorption of epidural morphine

In this letter from Drs B. Scheinin, R. Orko and P. H. Rosenberg, the legend to Fig. 1 gave the morphine plasma concentration incorrectly as 6 mg in 3 ml for the first patient group. The correct legend is:

Fig. 1. Plasma concentrations in the three groups of patients. ○, Morphine 6 mg in 30 ml; ●, morphine 6 mg in 3 ml epidurally; ▲, morphine 6 mg in 3 ml intravenously.

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........................19

To the Honorary Secretary,
Association of Anaesthetists of Great Britain and Ireland

I (full name).. offer my name as a candidate

BLOCK LETTERS

for (a) MEMBERSHIP (b) OVERSEAS MEMBERSHIP (c) ASSOCIATE MEMBERSHIP (d) JUNIOR MEMBERSHIP of the Association (*please delete categories not applicable*)*

I hold the following appointment(s) as an anaesthetist (*please state grade if in the National Health Service, and indicate the approximate proportion of time spent in the practice of anaesthesia*):

....

....

....

Qualifications (with dates)...

....

(Signed)...

Address...

....Hospital Tel. No:......................................

....Home Tel. No:...

We, being Members† of the Association, nominate as a candidate for MEMBERSHIP/OVERSEAS/ASSOCIATE/ JUNIOR MEMBERSHIP

....

1. (Signed)...

(NAME IN BLOCK LETTERS)

Address...

BLOCK LETTERS

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* See the reverse of this page for details of membership categories.

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Dr J. N. Lunn, Editor of ***Anaesthesia****, Department of Anaesthetics, University Hospital of Wales, Heath Park, Cardiff CF4 4XW, UK.*

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Type manuscripts on white bond paper, 20.3 × 26.7 cm or 21.6 × 27.9 cm (8 × 10½ in. or 8½ × 11 in.) or ISO A4 (212 × 297 mm) with margins of at least 2.5 cm (1 in.). Use double, and preferably triple, spacing throughout, including the references. Please do not use a dot matrix printer, particularly one with poor quality descenders or ascenders. Unseparated, fan-folded manuscripts may be returned to the author. *The manuscript* should consist of the following sections in this order each beginning on a new page: *title page*, *summary* and *key words*, *text*, *acknowledgments*, *references*, *individual tables*, and *legends for figures*.

Number pages consecutively, beginning with the title page.

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The second page should carry a summary of not more than 150 words. The summary should state the purpose of the study or investigation, basic procedures, main findings and their statistical significance, and the principal conclusions.

Do not use abbreviations except for units of measurement (e.g. mg, cm, etc.).

Key (*indexing*) *words*. Below the abstract, provide and identify as such, three to 10 key words or short phrases that will assist indexers. Use terms from the Medical Subject Headings list from *Index Medicus.* The Editor may modify these at proof stage to conform with agreed practice of certain other anaesthetic journals in the English language.

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(*continued overleaf*)

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The text of observational and experimental articles, case reports, etc. is usually divided into sections with the headings *Introduction*, *Methods*, *Results* and *Discussion*. Long articles will need subheadings within some sections to clarify their content.

Letters for the correspondence pages should be double spaced and prepared in accordance with the format in a recent copy of *Anaesthesia*.

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(i) CAPITALS across the centre of the page.

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These should be typed at the left-hand side of the page above the paragraph which they precede.

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Number references consecutively in the order in which they are first mentioned in the text. Identify references in text, tables and legends by arabic numerals. References cited only in tables or in legends to figures should be numbered in accordance with a sequence established by the first identification in the text of the particular table or illustration. Use double or treble spaced typing.

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Avoid using abstracts as references except those published in a recognised journal. 'Unpublished observations' and 'personal communications' may not be used as references, although references to written, not verbal, communications may be inserted (in parentheses) in the text. Include among the references manuscripts accepted but not yet published; designate the journal followed by (*in press*) in parentheses. Information from manuscripts submitted but not yet accepted should be cited in the text as (unpublished observations) in parentheses.

The references must be verified by the author(s) against the original documents.

Examples of correct form of references

Note: first and last pages in all references.

JOURNAL

Standard journal article—(List all authors)

Soter NA, Wasserman SI, Austen KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *New England Journal of Medicine* 1976; **294**: 687–90.

Corporate author

The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36**: 119–25.

Anonymous. Epidemiology for primary health care. *International Journal of Epidemiology* 1976; **5**: 224–5.

BOOKS AND OTHER MONOGRAPHS

Personal author(s)

Osler AG. *Complement: mechanisms and functions.* New York: Prentice-Hall, 1976.

Corporate authors

American Medical Association Department of Drugs. AMA drug evaluations, 3rd edn. New York: Publishing Sciences Group, 1977.

Editor, compiler, chairman as author

Rhodes AJ; Van Rooyen CE, *comps. Textbook of virology: for students and practitioners of medicine and other health sciences*, 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

Chapter in book

Weinstein L, Swartz MN. Pathogenic properties of invading micro-organisms. In: Sodeman WA Jr, Sodeman WA, eds. *Pathologic physiology: mechanisms of disease.* Philadelphia: W.B. Saunders, 1974: 457–72.

Agency publication

National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968—June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series* **10**: Data from the National Health Survey, No. 69) [DHEW publication No. (HSM) 72–1036].

OTHER ARTICLES

Newspaper article

SHAFFER RA. Advances in chemistry are starting to unlock mysteries of the brain: discoveries could help to cure alcoholism and insomnia, explain mental illness. How the messengers work. *Wall Street Journal* 1977 Aug 12: 1(col 1), 10(col 1).

Magazine article

ROUECHE B. Annals of medicine: the Santa Claus culture. *The New Yorker* 1971 Sept 4: 66–81.

TABLES

Do not include tables in the text. Start a new sheet for each table and space the material adequately. The author(s) name(s) should appear in the top right-hand corner.

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Type legends for illustrations double spaced with arabic numerals corresponding to the illustrations. When symbols, arrows, numbers or letters are used to identify parts of the illustrations, identify and explain each one clearly in the legend.

CONVENTIONS, ABBREVIATIONS AND STATISTICS

Statistics and measurements should be given in figures except that numerals one to nine should be in words if not followed by a measurement symbol (e.g. 'two patients' but '2.0 mg'). The *Système International* (SI) will usually be used except that vascular pressures will be recorded in mmHg and cmH_2O. Imperial measurements will not be used except in an historic context. The 24 hour clock will be used.

Contributors are advised to study the SI *Conversion Tables* provided in the January 1978 issue of *Anaesthesia, Units, Symbols and Abbreviations* (*A Guide for Biological and Medical Editors and Authors*) published by the Royal Society of Medicine, London W1M 8AE, and *Uniform requirements for manuscripts submitted to biomedical journals* (*British Medical Journal* 1979; 1: 532–5).

The statistical tests used in the report should be stated clearly. Results should include 95% confidence limits for the main findings and probability estimates.

LETTERS FOR PUBLICATION

Should be addressed to Dr J. N. Lunn, Editor of *Anaesthesia*, Department of Anaesthetics, University Hospital of Wales, Heath Park, Cardiff CF4 4XW, UK.

Letters should be typewritten on one side of the paper only and double spaced with wide margins. Copy should be prepared in the usual style and format of the Correspondence section. Authors must follow the advice about references and other matters above. The degrees and diplomas of each author must be given in a covering letter which must be signed personally by all the authors.

Correspondence presented in any other style or format may be subject to considerable delay and may be returned to the author for revision.

The editors regret that failure to comply with the above requirements may result in a delay in publication of accepted papers.

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